Friedberger & Fröhner's Veterinary Pathology

[Authorised Translation]

TRANSLATED BY

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WITH NOTES ON BACTERIOLOGY

BY

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PREFACE TO THE SIXTH EDITION.

It was not granted to Professor Franz Friedberger, the joint author of this text-book, to live to see the publication of this—the sixth—edition of our work. He passed away on December 17th, 1902, at Munich, in his sixty-third year, after a painful illness of many years' duration. In him Veterinary Science lost its greatest and best-known exponent. In clinical analysis he was, indeed, without a rival! To this must be added his rare devotion to duty as a teacher, his great scientific individuality, his typical love of truth (refined by his severe self-criticism) in the prosecution of his investigations and in his manner of offering the results to the public; and, lastly, his aversion, due to his lofty character, to any kind of quackery, self-advertisement, and the advancement of merely specious theories. This rare union of so many eminent qualities at once explains how he came to found his own—the Munich—school of science, which, with its numerous pupils, continues to flourish and to maintain its influence. Not every master, as we all know, is destined to create his school!

To that band of pupils of Friedberger belongs the joint author of this work and the editor of this present new edition. It will be of interest to the readers of this text-book and the friends of the deceased master to hear something as to its origin. In 1880 I took over the post of a clinical assistant at the then Veterinary School of Munich, and was brought into the closest relations with the resident professor, Professor Friedberger, whose methods of instruction and investigation appeared to me worthy of the highest admiration owing to his remarkable perspicuity and his strictly scientific methods. When in 1882 I was called to Stuttgart to take up a professorship there, the first suggestion was made of our publishing a scientific text-book on veterinary pathology. In the spring of 1884, when I had to go to Munich to attend Franck's funeral, the plan was definitely settled and the preliminary work taken in hand. The division of labour which was kept up for subse-

quent editions, was that Friedberger should collect and arrange all the literary material, whilst I was responsible for the actual text of the work. Besides, each of us contributed the results of his own personal experience, though I need scarcely add that the sum of clinical observations contributed by Friedberger in our literary union was far in excess of my own. A more perfect harmony than that which marked our collaboration could not have been possible. Friedberger, in his character of editor, permitted me the fullest liberty of description and expression, differences of opinion between us being ever most readily settled. No misunderstanding between us was possible, as perfect trust existed. I, for my part, in many cases bowed to the riper judgment of my senior, while he as frequently led me, with my younger and livelier moods, back into the smooth paths of the *via media*.

Friedberger, with his wonted, tireless industry, brought together and revised the material for the present—the sixth—edition until within a few weeks of his death. Thousands of notes, often with critical and sarcastic comments, lie before me as living evidence of his indefatigable zeal. How he rejoiced over every really important and really valuable addition to our work! On the other hand, it should be added that he bitterly denounced any worthless addition to our still youthful science, whether due to idle theories or to immature dilettantism, as the empiricism or presumption of many writers. To him any sort of presumption was so essentially antagonistic!

In the publication of this new edition I have striven to continue the work in the spirit of Friedberger. The reader will find that his groundwork has been religiously preserved, whilst the investigations of the past four years have been fully noted. Strict limits, having regard to the enormous accumulation of new material on the subject of tuberculosis, have been observed, with a view to restrict within reasonable proportions the bulk of the work.

EUGEN FRÖHNER.

Berlin, April, 1904.

PREFACE TO ENGLISH EDITION.

WHEN my late husband, Captain M. H. Hayes, brought out the first edition of his English translation of this work, he divided it into two volumes termed respectively, "Infective" and "Non-Infective" Diseases. I have thought it better to discard this arrangement, and to adhere strictly to the German text and the division of the volumes and chapters throughout the entire work. I have the pleasure now to submit to the public an exact, authorised translation of the sixth edition of Professors Friedberger and Fröhner's Veterinary Pathology.

I have carefully gone over the English of the entire work with the German text at hand, submitting it then to Mr. William Hunting, F.R.C.V.S., who has taken a deep interest in it, and has devoted much of his valuable time to its revision.

As we see in his preface to the present German edition, Professor Fröhner announces the sad death of his friend and co-worker Professor Friedberger, who died at Munich after a long and painful illness. The Veterinary Profession has thus lost in him one of its most prominent and famous representatives.

I have to thank many kind friends for their help on this work, especially Mr. W. Hunting, F.R.C.V.S., for revising all the proofs and for several notes which he has added, and Professor R. Tanner Hewlett, M.D., F.R.C.P., M.R.C.S., D.P.H., for writing his *Notes on Bacteriology*. As this work is intended for English veterinary surgeons and students, I have deemed it best to give all the weights and measures in English.

ALICE HAYES.

The Bungalow,
Crick, Rugby, May, 1908.

WEIGHTS, MEASURES AND DEGREES.

As the metric system, which has been adopted by Continental scientific men, is not generally used by veterinary surgeons in English speaking countries, the following tables are given. Readers of this book should bear in mind, that when the word ***gramme*** is employed in connection with fluids, it means a cubic centimetre (c.c.), because that measure of pure water at 4° C. (39·2° F.), weighs a gramme. In prescriptions, when numbers are given without any allusion to weights or measures, grammes are meant.

RELATION OF METRIC WEIGHTS TO ENGLISH WEIGHTS.

1 Milligramme	=	0·015432 grains.
1 Centigramme	=	0·15432 ,,
1 Decigramme	=	1·5432 ,,
1 Gramme	=	15·432 ,,
1 Kilogramme	=	2 lbs. 3 oz. 120 grains (nearly).

RELATION OF METRIC FLUID MEASURES TO ENGLISH FLUID MEASURES.

1 Cubic centimetre (c.c.)	=	17 minims (nearly).
1 Litre	=	1·76 pint (nearly).

RELATION OF METRIC MEASURES OF LENGTH TO ENGLISH MEASURES OF LENGTH.

1 Millimetre	=	0·03937 inches.
1 Centimetre	=	0·3937 ,,
1 Decimetre	=	3·937 ,,
1 Metre	=	39·37 ,,

RELATION OF CENTIGRADE DEGREES TO FAHRENHEIT DEGREES.

A difference of 1° C. is equal to that of $1{\cdot}8^\circ$ F.; but their respective zeros are not the same. For instance, the freezing point of water is represented by 0° C., according to the metric system; and by 32° F., according to the popular English idea. Hence, to convert Centigrade degrees into Fahrenheit degrees, we have to multiply them by 1·8 and add 32. For instance, if we wish to find how many Fahrenheit degrees are in, say, 40° C., we can do so, by the following equation :

$$40^\circ \text{ C.} = 40 \times 1{\cdot}8^\circ \text{ F.} + 32^\circ \text{ F.} = 104^\circ \text{ F.}$$

If the required conversion is that of -20° C., the equation would be

$$-20^\circ \text{ C.} = -20 \times 1{\cdot}8^\circ \text{ F.} + 32^\circ \text{ F} = -4^\circ \text{ F.}$$

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bodies. Among chemical irritants in the case of herbivora, there are a great number of poisonous plants, such as aconite, hellebore, ranunculus, euphorbia, daphne, colchicum, corn-poppies, tobacco, corn-cockle, digitalis, yew, spotted hemlock, water-hemlock, fool's parsley, rhododendron, charlock, etc. Catarrhal stomatitis may be produced by the inadequate dilution of certain therapeutic agents, such as electuaries containing tartar emetic; solutions of corrosive sublimate; ointments containing arsenic, chloride of zinc and other substances which can be licked off; and solutions of caustics and acids. Pungent spices (pepper for instance), and the skins and roots of black radishes (*raphanus sativus*) produce a similar effect. Thermal irritation is caused by the consumption of hot food and drink, or by the scalding of the mucous membrane with hot draughts.

A further cause of catarrhal stomatitis is the ingestion of fodder which has become infected by blight-fungi (*uredineæ, pyrenomycetes*). These fungi frequently produce not only a catarrhal, but also a vesicular and ulcerous inflammation. The "rape-destroyer" (*polydesmus exitiosus*), often causes severe stomatitis in cattle and sheep. *Puccinia graminis* (common grass and corn blight), *puccinia arundinacea*, and the uromyces fungus, which lives on Swedish clover, have a similar action. Among the rusts, bunt (*tilletia caries*), which was formerly called *uredo fœtida*, or stinking rust, plays a prominent part. Mould-fungi (as in mouldy oats) and mildew (*erysibe communis*) are not potent producers of this disease. These fungi have essentially a chemical action (toxine).

The consumption, along with the food, of the hairs of caterpillars is a special cause of stomatitis. The hairs of the *bombyx s. cnethocampa processionalis*, and of the hawthorn caterpillar (*porthesia chrysorrhœa*). Besides the mechanical irritation, it is probable that a chemical poison (enzyme or formic acid) is also in action. For instance, Kösters saw 52 artillery horses ill on account of their green food having become tainted by hawthorn caterpillars. In a similar way, plant lice (*aphis*) produce stomatitis in herbivora.

Apart from causes which have a directly irritating effect on the oral mucous membrane, stomatitis occurs in all the domestic animals, and especially in horses, from unduly prolonged abstinence from food. This form of the disease is particularly well marked in digestive disturbances, in which cases the cast-off epithelial masses, which are usually removed during the con-

sumption of food, become decomposed, and consequently set up inflammation of the buccal membrane. The same remark applies to chronic general diseases of a febrile type, in which there is the further aggravating circumstance that the digestive secretions (saliva in this case) become decreased in quantity and morbidly changed, which fact increases the liability of the desquamated epithelium to become decomposed. A special form of stomatitis (stomatitis mercurialis) is found in mercurial poisoning. Sometimes catarrhal stomatitis is developed by the spreading of an inflammation of the mucous membrane from the neighbouring parts, as, for instance, during the course of pharyngitis. Besides the purely traumatic influence of teeth which are cast off during the stomatitis that occurs in teething, we have to take into consideration the hyperæmia and inflammation of the gums (*gingivitis*). We have no certain knowledge as to the possibility of stomatitis being developed from a chill.

Symptoms.—In all our domestic animals the symptoms of simple catarrhal stomatitis vary greatly according to the degree of the inflammation. In very slight cases we find only dryness and heat of the mucous membrane; but later on, a pappy, greyish-white, frothy coating appears, especially on the dorsum linguæ. Such cases of slight stomatitis are most frequently observed as accessory symptoms of general febrile diseases. The so-called "coated" tongue is a result of an increased production of epithelium, due to the catarrhal condition, and to the fact that the mechanical removal of the epithelium becomes checked, in consequence of the diminution in the amount of food consumed. It is also, in part, due to the increased dryness of the individual layers of the epithelium, owing to a decrease in the amount of secretion due to a state of fever. The mucous membrane of the lips and cheeks may also become congested. The portion of the hard palate which is behind the incisor teeth is more or less swollen in consequence of the venous congestion of the very vascular submucosa, as in lampas of horses. There is also a mawkish, sweet smell from the mouth. In severe cases of catarrh there is increased redness and swelling of the mucous membrane, and especially of that of the lips, cheeks and gums. Also, an increased production of saliva and mucus causes viscid glairy strings to drip from the corners of the mouth ; and more or less frequently, very abundant saliva is either beaten into froth, or flows in an uninterrupted stream from the lips

(slobbering). The prehension of food is somewhat suppressed. If the epithelium be very abundantly cast off, the dorsum linguæ gets thickly coated, and assumes a dirty green, or brown colour, on account of its becoming soiled by particles of food; and a sweetish putrid smell issues from the mouth. In cats, the papillæ of the tongue, which resemble little horny teeth, appear strikingly white or yellowish white, in contrast to the bright red edges of the tongue. We sometimes observe in cattle and pigs hypertrophy of the epithelium of the inflamed cone-shaped papillæ, which has been erroneously regarded as thrush (the so-called "brush" on the tongue of cattle). In birds catarrhal stomatitis manifests itself by the horny covering of the tongue becoming dry and opaque, and consequently laymen often make the mistake of thinking it is a pathological membrane, and wrongly pull it off.

The erosions which occasionally occur during the course of simple catarrhal stomatitis, are small and superficial losses of substance of the mucous membrane, which usually heal rapidly, and therefore differ essentially from true necrotic ulcers. The same remark applies to the very rare ulceration of the mucous glands, which we have observed on a few occasions in horses during the course of strangles and rickets, and which were formerly known as *stomatitis folliculosa*. It should be regarded as a complication of catarrhal stomatitis. It begins with the formation of small grey or dun-coloured nodular elevations which are surrounded by a red area. The nodules appear as a consequence of the excretory ducts of the numerous glands becoming plugged up. Further, under certain conditions in cattle, a simple catarrhal stomatitis may raise a suspicion of foot-and-mouth disease (*cp. Stomatitis aphthosa*).

Therapeutics.—Catarrhal stomatitis is a very mild affection, which, as a rule, can be cured by the removal or exclusion of the irritating causes. A proper method of treatment greatly accelerates recovery, which is very desirable, because animals that are suffering from this disease do not feed well, consequently lose flesh and are incapable of hard work. The simplest treatment consists in the frequent irrigation of the mouth with fresh and pure spring water. Water acidulated with vinegar, is an old domestic remedy for stomatitis. Such a mouth douche is generally prepared by adding a large tablespoonful of common salt and a quarter of a pint of vinegar to a quart of spring water. More obstinate

cases may be treated with disinfecting solutions, such as 1 to 4 per cent. watery solutions of chlorate of potash or boric acid, and in severe cases, $\frac{1}{4}$ to 1 per cent. of creolin, with which the mucous membrane may be irrigated or painted. Solutions of permanganate of potash are less reliable, because they are feeble disinfectants and have a disagreeable taste. Astringent solutions—such as $\frac{1}{2}$ to 1 per cent. of alum or tannin, decoctions of oak-bark, garden sage leaves (*salvia officinalis*), etc.—are indicated when there is a very large secretion of saliva and mucus. Internally we may, at the same time, give common salt, sodium bicarbonate, sodium sulphate, etc.

As regards feeding, we should bear in mind that in all severe cases of oral catarrh only fluid or soft food should be given. If unwholesome food is the cause of the disease, a change of diet should of course be made.

Salivation (*Ptyalism*).—Formerly salivation was regarded as an independent disease, but it is merely a symptom of various diseases. It is generally caused by the presence of stomatitis, and also by foreign bodies in the oral cavity, pharynx, and œsophagus ; by pharyngitis, and by obstruction in, or paralysis of, the organs of deglutition. Paralysis of the pharynx and œsophagus occurs in rabies, during gestation, in diseases of the brain, and poisoning, particularly by bunt (*tilletia caries*), or other fungi in spoilt food. Deglutition is also almost impossible in tetanus. Salivation is observed, particularly with dogs, in diseases of the stomach and intestines, but seldom in helminthiasis. It appears in a few cases of parotitis, and, by reflex action, in diseases of the uterus and ovaries. Pilocarpine, arecoline, mercury, iodine and morphia are the chief drugs that excite salivation. The cause of salivation often remains obscure. For reducing the secretion of saliva, we may try atropine or hyoscyamine by injecting it subcutaneously in horses and cattle in $\frac{1}{8}$ grain doses, and in $\frac{1}{100}$ grain doses in dogs. Saliva has an alkaline reaction in health, but is found to be acid in various human diseases, such as pleuritis, encephalitis, rachitis, dyspepsia, and diabetes. In order to test the accuracy of this statement with respect to animals, we have made many experiments in cases of disease, but have never found an acid reaction of the saliva. The occurrence of an acid reaction of saliva in man may be accounted for by the formation of acid in the mouth caused by decomposing particles of food, hollow teeth, etc.

2. STOMATITIS APHTHOSA (*vesicularis*). SPORADIC APHTHÆ OF THE ORAL MUCOUS MEMBRANE.

Nature and Causes.—In veterinary medicine the name of aphthæ is given to vesicles on the oral mucous membrane, which are produced by an accumulation of serum under the epithelium of the mucous membrane, and, consequently, stomatitis aphthosa is identical with vesicular stomatitis. A typical stomatitis aphthosa is found in foot-and-mouth disease, which is a specific infective disease that is discussed in Volume II. of this book. There is a contagious and enzootic aphthous stomatitis which appears occasionally in horses, cattle and sheep, and which is entirely independent of foot-and-mouth disease. It has been known from remote times as "sporadic aphthæ," and its causes are most probably fungi which attack forage, particularly those that infect Swedish clover (*uromyces*) and rape (*polydesmus exitiosus*). These and other infective fungi produce in some cases a catarrhal, in others an aphthous, and sometimes even an ulcerous or croupy stomatitis. Also, the hairs of caterpillars and other substances may set up an aphthous stomatitis by intense irritation, or by individual predisposition. In some instances, the cause remains unknown; but can, as a rule, be found in a changed condition of the food.

Symptoms.—The phenomena of aphthous stomatitis consist of the formation of vesicles on the mucous membrane of the lips, cheeks, tongue and gums, which vesicles are filled with clear serum, and are either isolated or massed in large numbers. They usually burst in a very short time and become changed into congested sores (erosions) which quickly heal. They vary in size from that of a pin head to that of half a crown. Besides the vesicles, there are usually symptoms of catarrhal stomatitis, with redness and swelling of the mucous membrane and salivation. We may sometimes notice a "smacking" noise during the movements of the lower jaw, similar to that which takes place in foot-and-mouth disease. After the bursting of the vesicles, the mucous membrane of the mouth may remain painful for some time. There is failure of appetite, and simultaneously with the appearance of the vesicles, we may sometimes find a feverish rise of temperature, which rapidly subsides. It occasionally happens, particularly in cases

of "clover disease," that, besides the inflammation of the mucous membrane, an erysipelatous dermatitis breaks out on the head, udder, limbs and other parts of the body. The nature of the disease is generally benign and its course rapid.

Differential Diagnosis.—Sporadic aphthæ may be readily confounded with foot-and-mouth disease. Experience teaches that a prompt and exact differentiation is not always possible, especially when several animals become simultaneously affected in consequence of the consumption of the same food, and when fever is also present, or when the ingestion of the fungi is followed by gastric symptoms, or by symptoms of a general mycotic intoxication. The fact that the disease produced by these fungi cannot be transmitted to healthy animals will be decisive, and, consequently, in doubtful cases inoculation experiments should never be omitted. Also, the coronet of the hoofs in sporadic aphthæ is, as a rule, free from aphthous lesions, and the aphthous affection of the oral mucous membrane usually heals more rapidly than in foot-and-mouth disease, in which the vesicles are generally larger.

Therapeutics.—The treatment of aphthous stomatitis consists in the frequent rinsing out of the mouth with fresh spring water, avoidance of rough forage, and in the use of disinfectant and astringent lotions (boric acid, potassium chlorate, creolin, salicylic acid, and alum, tannin, sulphate of iron, etc.). Also, the mixture of vinegar, common salt and water, to which allusion was made (p. 4) when discussing catarrhal stomatitis, may be used as a domestic remedy.

In 1897, Theiler observed a contagious stomatitis in horses and mules in South Africa—the "blauwtong" of the Transvaal Boers. This form, which had already been noticed in Cape Colony in 1884 by Hutcheon, is distinct both from foot-and-mouth disease and from *stomatitis pustulosa contagiosa.*

3. STOMATITIS ULCEROSA.

Definition.—Stomatitis ulcerosa consists in a necrotic inflammation of the oral mucous membrane, which spreads to a varying extent, and which attacks the gums more frequently than other parts. Anatomically we may regard it as a very severe form of the catarrhal and aphthous process, which manifests itself by the formation of deep ulcers on the mucous membrane.

Etiology.—Apart from traumatic, thermal and chemical causes, stomatitis ulcerosa is found most frequently in delicate, pampered, weak and anæmic dogs of the smaller breeds, and in cats. This disease is sometimes preceded by debilitating affections, such as gastro-intestinal catarrh, rachitis, and distemper. Ulcers, which appear in such cases, were formerly looked upon as scurvy (an erroneous idea); because similar ulcers may be seen in otherwise healthy animals, without any hæmorrhage into the mucous membrane, as in scurvy. The cause of ulcers in non-scorbutic stomatitis is probably abnormal vulnerability, and low power of resistance of the mucous membrane from individual idiosyncrasy, in combination with an infection. Carious teeth, or teeth which are thickly covered with tartar, are very often the starting point of the affection, on account of an infectious inflammation of the gums being set up by them. The statistics of the Berlin canine hospital have shown that stomatitis ulcerosa forms about one-half per cent. of all the cases of diseases of dogs at that institution.

Stomatitis ulcerosa is sometimes caused in horses, cattle and sheep by the consumption of food infected by fungi, chiefly pyrenomycetes and uredineæ, which give rise to ulcerous inflammation of the mucous membrane. Thus stomatitis ulcerosa has occurred enzootically in horses from eating Swedish clover, and in lambs from feeding on infected rape-cakes. In horses, ulcers as large as a shilling have been found upon the mucous membrane of the lips (Ugrai) after feeding with hay containing much bristle-grass (*setaria viridis* and *verticillata Beauv.*). In Batavia, the mould-fungus (*hyphomycosis destrueus equi*) has been shown to give rise to a severe form of ulcerous stomatitis in horses (De Haan). Certain bacteria have also been regarded as pathogenetic factors in this disease (Rivolta).

As a secondary symptom, we find ulcers of the oral mucous membrane in foot-and-mouth disease of ruminants and pigs; in stomatitis pustulosa contagiosa of horses; in avian diphtheria and in mercurial poisoning.

Symptoms in Dogs.—The gums in the neighbourhood of certain teeth at first become dull red, a condition which is observed particularly near the incisors and canines, sometimes also round the molars, on both jaws, and on one or both sides; the portion of the gum surrounding the neck of the tooth is also swollen. Even in one or two days there is a well-marked increase in the swelling of the affected tissue, which is

of a dark red, brown red, or purple colour, and looks spongy, swollen and loosened. It has receded a little from the respective teeth, and bleeds on the slightest pressure. Later on, the affected tissue becomes discoloured, this change being initiated by the swollen part which is next to the teeth, and which assumes a tint varying from greyish green to greyish yellow. As a consequence of the cellular infiltration and necrosis of the mucous membrane, we have the necrotic crust, which at first is friable, and, later on, greasy and pulpy. A spontaneous or forcible removal of this mass reveals a large ulcer, which is usually about one-sixteenth of an inch deep, of varying size, and sometimes circular when at the canine teeth. Its edges wind in and out in an irregular manner, are usually raised in the form of a wall, hard to the touch, and very hyperæmic. The base of the ulcer is uneven and discoloured. At the same time, there is considerable slavering, and glairy, ropy, brown-red mucus, which is often mixed with blood, flows from the corners of the mouth, and soils all the adjoining parts. Such masses of mucus are also found in large quantities in the mouth itself, and are discharged when it is forcibly opened : a proceeding from which dogs generally try to escape, as it hurts them. The discharge, the entire oral cavity and the expired air all have a very fœtid, nauseous and putrid smell, which often taints the entire neighbourhood of the patient. Besides the circumscribed localised ulcers, the symptoms of simple catarrhal stomatitis are also present.

The above described phenomena of inflammatory hyperæmia, swelling, cellular infiltration and necrotic disintegration of the mucous membrane appear not only on the gums in the immediate neighbourhood of the teeth, but also on other parts of the interior of the mouth, as, for instance, on the anterior surface of the gums, lips, corners of the mouth, less frequently on the cheeks, and very rarely on the lower surface of the tongue. The ulcers on the lips frequently occupy positions directly opposite those on the edges of the gums close to the teeth, and consequently we think that they arise, in such cases, from auto-infection. In other instances the arrangement of the ulcers suggests that infection was conveyed by the lymph passages. We also meet with cases in which the morbid process in no way affects the teeth or the mucous membrane in their immediate neighbourhood ; but begins on other parts of the gums, on the lips, or even on the corners of the mouth, and occasionally spreads to other portions of the common integument.

Process.—In favourable cases and in young animals, the ulcers enlarge only to a moderate depth and extent. The general health is but little disturbed, fever is absent or remains very low (about 103° F.), the appetite is surprisingly good, and after the ulcerative process has stopped and the ulcers have become clean, these lesions usually heal very quickly, even as soon as eight or ten days. Such cases are the most frequent.

In other instances, particularly with old animals that are suffering from another complaint, the infiltration and ulcerative disintegration of the affected tissue progress rapidly. The ulcers become deep and sinuous, the implicated teeth get loose and can be easily pulled out; their roots are covered with a grey, stinking, pulpy substance (the destroyed alveolar periosteum), and caries and necrosis may supervene. Sometimes a fistula is formed between the mouth and nasal passages, particularly on the site of the canine teeth, with subsequent suppurative rhinitis. The affected parts of the lips and cheeks are hard and tense to the touch, are more or less œdematously swollen, and there is an inflammatory swelling of the adjoining lymph glands. In these cases the general health is disturbed, and recovery may not take place for several weeks or even months.

If the course of the disease be unfavourable, we may often observe, during freshly appearing eruptions, that persistent fever of a varying character sets in; the pulse becomes more and more frequent and smaller, the appetite is entirely lost, diarrhœa makes its appearance, the animal gets very weak and prostrated, and finally dies from septicæmia.

Symptoms in Herbivora.—When stomatitis ulcerosa of horses, cattle and sheep is produced by fungi, it manifests itself by ulceration, swelling, congestion and pain in the mucous membrane of the mouth and lips, and by copious slavering. In severe cases, the oral mucous membrane, especially of the gums, palate and sides of the tongue, exhibits circumscribed, diphtheria-like changes in the form of grey or grey-brown raised spots, which vary in size from a pea to half a crown, and which, after removal of the crust-like scab, show a deep red, and sometimes briskly granulating ulcerous surface. We may not unfrequently notice severe general disturbance (mycotic poisoning), which is specially well marked in young animals, and which has been described by Berndt in connection with lambs. He found that the principal symptom was an acute inflammation of the nose and mouth, which almost always ended in death. The general symptoms consisted in great debility, staggering gait, accelerated and difficult breathing, and faint short cough. The mucous membrane of the gums and the tongue showed numerous erosions, which were also present on the visible part of the Schneiderian membrane,

where they were covered by a thick brown crust. On *post-mortem* examination, the mucous membrane of the nose, mouth, pharynx and larynx were found swollen and partly eroded; the lower edges of the posterior lobes of the lungs were hard and dark red; their section-surfaces, from which an ichorous fluid flowed, were granulated; and the divided bronchi contained a frothy fluid, which could easily be pressed out. Microscopic examination showed that numerous brown, ramified threads and chains of spores of *polydesmus exitiosus* existed between the epithelial cells of the oral and nasal mucous membranes. These lambs had been fed on rape cakes, in which spores of the ascomycetes were found. After the feeding on these cakes was stopped, no more cases of infection occurred. Rivolta has described the appearance, in young lambs, of a similar very malignant epizootic stomatitis ulcerosa, which was complicated with conjunctivitis, pneumonia and hepatitis. Gips, Bertsche and others have reported similar cases of stomatitis in lambs.

Therapeutics.—The treatment of stomatitis in dogs is essentially surgical, and consists in careful and repeated disinfection of the affected mucous membrane. As mild antiseptics, such as boric acid, salicylic acid and potassium chlorate, are not generally effective, we would recommend, from the very first, the application of the strongest disinfecting agents, of which creolin is one of the best. By the use of a 1 to 2 per cent. watery solution of creolin we have obtained surprisingly good and rapid results. The solution of creolin should be carefully applied with a brush four or five times a day to each ulcer. A 1 to 4 per cent. solution of aluminium aceto-tartrate or zinc chloride; solutions of perchloride of mercury (1 to 1,000), sulphate of copper and nitrate of silver (1 to 2 per cent.); and touching the ulcers with caustics are efficacious means. When using the last-mentioned metallic salts, great care should be taken that only the ulcers are touched, and that none of the fluid is swallowed. We have sometimes observed favourable results from the application, with a brush, of alcoholic remedies, such as tincture of myrrh and aloes. The early and free extraction of carious and loose teeth is of essential importance for recovery.

PAROTITIS (*Inflammation of the Parotid Gland*).

Occurrence.—Although this disease is comparatively frequent among mankind, in whom it is commonly known as "mumps," it has received but little attention in veterinary literature, which neglect is explained by the fact that it very seldom occurs among our domestic animals. It is particularly rare in dogs. Out of 70,000 cases of sick dogs in the Berlin

hospital, during nine years, there were only six cases of parotitis. But, according to certain French authorities, the mumps of human beings is distinctly transmissible to dogs, and can be given by one dog to another (Busquet and Boudeaud). And Bissauge states that in the neighbourhood of Orleans he repeatedly noticed a general attack of parotitis in cattle at a time when there was an epidemic of mumps among children.

Etiology.—This disease may be traced to several causes :—

1. *Traumatic parotitis*, produced by mechanical injuries, which should be treated surgically.

2. *Primary idiopathic parotitis*, which is caused by a miasmatic contagium, and appears epizootically. To this form we may ascribe the cases observed by Aruch in horses, by Franze in goats, by Hertwig in dogs, and by Schüssele in dogs and cats, which were epizootic and analogous to epidemic mumps in man. As regards the pathogenesis of this chief form of parotitis, it is possible that the contagium is carried by the blood to the parotid gland, or, what is more probable, that it reaches it from the oral mucous membrane *via* Stenon's duct.

3. *Parotitis caused by the extension of a neighbouring inflammatory focus*, as, for instance, by the presence of an impacted salivary calculus, or by inflammation of the buccal membrane spreading through the salivary duct, as in a case described by Toussaint, in which the inflamed salivary duct became dilated into the shape of a funnel. The morbid process in strangles, angina and distemper, may extend to the parotid gland in a similar way.

4. *Metastatic parotitis* during the course of pyæmia and septicæmia, and, as stated, in strangles, pleuro-pneumonia and distemper.

5. *Parotitis caused by the immigration of actinomyces bovis* (Parotitis—actinomycosis) is a secondary symptom of actinomycosis, which is an infective disease, and occurs epizootically among cattle in certain districts. This parotitis differs from the other forms by being chronic.

Stockfleth describes, under the heading of "Chronic Inflammation of the Salivary Glands," in his book on surgery, a disease which is without doubt actinomycotic parotitis. He explains that the inflammation occurs particularly in cattle, which graze on low-lying meadows near the seashore, and are fed with hay produced on these meadows. The parotid gland is much more frequently affected than the submaxillary or sublingual. The course of the disease is very slow. The gland shows, hard, uneven,

painless swellings, which gradually increase in size, and usually form abscesses later on. A secondary swelling of the lymph glands nearly always supervenes. Finally, the swollen gland presses on the upper part of the pharynx and larynx and causes difficulty in deglutition and respiration. The administration of medicines is entirely useless, and a cure can be expected only by excision of the swollen gland. For further information on actinomycosis and glossitis, see Vol. II.

Symptoms.—In the three forms of parotitis (idiopathic, metastatic, and that spreading from a neighbouring focus) which we are dealing with here, there is a more or less well-marked swelling of the parotid gland. In idiopathic parotitis this swelling spreads more uniformly over the gland than in the other two varieties, and seldom suppurates. In the other two forms it is more circumscribed, and is usually followed by suppuration of the implicated lobules. It may also extend to adjoining parts. Thus, Franze observed in an epizootic attack of this disease in goats, that the upper and lower jaw and the submaxillary glands were swollen and inflamed, and consequently the animals were unable to eat or drink. Aruch saw during eighteen months only one unilateral case among seventy horses which were suffering from parotitis and maxillitis. At the same time, there were symptoms of stomatitis. According to Bissauge, the attack commences in cattle with stiffness of the neck, loss of appetite, slavering and difficulty in swallowing. After three or four days, an inflamed and painful swelling of the glands makes its appearance, generally upon one side only and accompanied by a high temperature (104° F.). Slight mastitis, orchitis, and abscesses in the parotid occurred as complications now and then. According to Busquet and Boudeaud, dogs showed the following symptoms upon about the third or fourth day of the period of incubation : swelling of the parotid and submaxillary glands, with local œdema of the skin, accompanied by depression, loss of appetite, heightened temperature, sneezing and cough. The attack reaching its height after the twelfth day. We have never observed salivation during parotitis.

Differential Diagnosis.—Parotitis has probably been frequently confounded with inflammation of the subparotid lymph glands during strangles and pharyngitis, which mistake could certainly be avoided by a knowledge of the anatomical position of these lymph glands, and of the history of the case. We may also have to consider swelling of the guttural pouches,

affection of veins caused by a fistula produced by phlebotomy, simple œdematous or phlegmonous inflammation of the skin and, especially, of the subcutaneous connective tissue which covers the parotid gland, and various kinds of swellings of the parotid gland.

Therapeutics.—The treatment of non-suppurating parotitis consists in the application of moist heat (Priessnitz's bandages) and inunction with ointment of camphor, creolin, carbolic acid, or iodoform. In chronic cases more active means, such as tincture of iodine, biniodide of mercury ointment and cantharides ointment may be tried. For actinomycotic parotitis of cattle, we would recommend the internal administration of potassium iodide, from 1 to 4 drams daily, or what is equally effectual and far less expensive, from 5 to 7 grains of biniodide of mercury, dissolved in water by means of an equal quantity of potassium iodide. Abscesses should be opened as early as practicable. Any resulting salivary fistulæ should be treated surgically.

PHARYNGITIS AND PHARYNX-ANGINA.

General Remarks.—The name of angina (inflammation of the throat) has for a long time been applied generally to inflammation of the mucous membrane of the pharyngeal cavity and its adjoining parts, especially the larynx; because an exclusive affection of the pharynx is rare, particularly in horses. Sometimes pharyngitis is best marked in this inflammation of the throat; at other times, laryngitis. Hence it is advisable to divide this complaint into pharynx-angina and larynx-angina, according as pharyngitis or laryngitis is more in evidence. We shall here discuss only pharynx-angina, and will put larynx-angina among the "Diseases of Respiration" (Vol. II.).

In the smaller domestic animals we are able to sub-divide pharynx-angina into separate local affections, according to its espective sites on different parts of the pharynx, as, for instance inflammation of the soft palate, tonsils, and true pharynx; but in large animals, like horses and cattle, we cannot do this without complicated instruments. The term pharynx-angina, therefore, signifies in veterinary medicine, a more or less extensive inflammation of the mucous membrane of the pharyngeal cavity, including the soft palate and tonsils, in which case,

usually, though in a subordinate manner, the mucous membrane of the larynx, upper part of the trachea, mouth, nose and rarely that of the Eustachian tubes and guttural pouches become involved.

The difficulty of clinical examination of the larger animals renders it hard to determine the character and form of the disease. The differentiation of catarrhal, phlegmonous, suppurative, hæmorrhagic, croupy, diphtheritic, lacunous and necrotic forms can be made with ease only in the smaller animals. We can, however, make a fairly exact examination of the mucous membrane of the pharynx of horses by means of Polansky and Schindelka's rhino-laryngoscope, which is too complicated for general practice, and cannot be used with cattle. We may, therefore, as a rule, only indirectly infer the presence of one of the above-mentioned forms of pharyngitis, as, for instance, the catarrhal or phlegmonous variety, from the difference in the intensity of the symptoms, secondary implication of the lymph glands, fever, duration of the disease, etc. The croupy form can be recognized only by the ejected or coughed-up croupy membranes, and even then we are unable to decide whether the croupy shreds come from the larynx, or from the pharynx. We shall, therefore, try to give a general sketch of pharyngitis, without laying stress on the classification of the above-mentioned special forms.

Etiology.—Pharyngitis occurs sporadically or epizootically in all the domestic animals, but most frequently in horses, dogs and pigs; after them, in cattle and cats; seldom in sheep and birds, except in cases of avian diphtheria. Among general causes, we may assume the influence of individual predisposition in the domestic animals, although it is not so well marked as in man. The true nature of this predisposition in both animals and man is unknown. It is, however, certain that young animals and those which are weak, pampered, and habituated to warm abodes, frequently suffer from this disease, although old and robust ones are not entirely immune. The following are the chief special causes of pharyngitis:—

1.—Mechanical, chemical and thermal irritants which directly affect the mucous membrane of the pharynx. Under injuries thus produced, we may include bruises and lacerations of the mucous membrane caused by sharp meat-bones, fish-bones, thorns, needles, rough, coarse and prickly food, awns of barley, careless manipulation of the oral cavity (as, for instance, unskilful filing of the teeth), etc. Larvæ of gad-flies which have attached themselves to the mucous membrane of the pharynx, may produce a more or less chronic pharyngitis. Also tumours that project into the pharyngeal cavity may mechanically occasion catarrhal inflammation of the mucous membrane, as

we find in the case of actinomycomata and tubercular new-growths, both of which are common ailments in cattle. The chief chemical irritants are the poisonous plants mentioned in connection with catarrhal stomatitis (p. 2); drugs which, usually from inaccurate compounding, cause erosions of the mucous membrane, as, for instance, tartar emetic in an undissolved condition, croton oil in too concentrated solutions, ammonia not sufficiently diluted, metallic salts, arsenic, phosphorus, etc. Fodder which contains pyrenomycetes and uredineæ sometimes give rise to both stomatitis and pharyngitis. Hot drinks which scald the mucous membrane, hot food, and the inhalation of steam may also set up angina.

2.—Chills, especially local chills in the pharynx, caused by the inhalation of cold air when north or north-east winds prevail, by the drinking of very cold water, the consumption of food covered with hoar frost, etc., may act as local irritants in setting up pharyngitis. We have also to consider the effect of cold in a wider sense, as, for instance, chill of the surface of the whole body, in consequence of a sudden change of weather, rain, or any great fall of temperature. Although it is certain that too much importance has been attached to the influence of chill in the production of disease, yet with our present state of knowledge this factor cannot be entirely ignored, especially in the case of pharyngitis.

3.—*The spreading of inflammation from neighbouring organs to the mucous membrane of the pharynx.* Stomatitis, as well as rhinitis and laryngitis, may cause pharyngitis by spreading. As pharynx-angina is the most common symptom in strangles, the two diseases are often mistaken for one another. It also occurs as a complication in foot-and-mouth disease, rabies, distemper, pleuro-pneumonia, petechial fever, anthrax, acute and chronic glanders, tuberculosis, etc.

4.—Infection. Many cases of pharynx-angina appear to be caused by a specific infection, by which the disease assumes a contagious character. Outbreaks of epizootic angina certainly bear out this supposition. In this respect we must be very careful, especially with horses. The fact that strangles frequently exhibits symptoms of pharyngitis, has certainly resulted in many mistakes being made between this disease and epizootic strangles. In any case, however, epizootic pharyngitis among horses, accompanied by suppuration, must always be regarded as strangles (pyogenetic strangles streptococcus). Influenza among horses sometimes appears as infectious laryngo-

pharyngitis. Infectious epizootic pharyngitis, which may lead to metastatic pyæmia, sometimes occurs among puppies during the first few weeks after birth. An infectious diphtheria, identical with that of human beings, does not seem to occur among domestic animals. But contagious diphtheritic pharyngitis is by no means uncommon among swine, and sporadic angina produced by the necrosis bacillus frequently occurs among cattle (*cp.* Vol. II. Diphtheria).

The attachment of bots on the pharyngeal mucous membrane must not be readily accepted as a cause of pharyngitis, because we know from experience that these larvæ are frequently found on this membrane, as they also are in the stomach, of perfectly healthy horses. The case of fatal pharyngitis in a remount, described by Lichmann, in which 14 bots were observed on the pharyngeal mucous membrane, cannot be referred to these larvæ; because the animal was one of several horses which were suffering from severe inflammation of the throat.

Verminous Pharyngitis.—In the case of a Japanese cavalry horse, which had died under symptoms of pharyngitis, Tokishige found a tumour-like affection of the mucous membrane of the pharynx, showing nodules in which microscopic examination revealed minute nematoid worms with numerous eggs. He gave these thread-like worms the name of Dispharagus reticulatus.

Anatomical Conditions.—In mild cases of catarrhal pharyngitis the mucous membrane of the pharynx is either diffusely congested or shows red spots; it is swollen, and covered with glairy or purulent mucus, which may be streaked with blood. It sometimes displays long narrow erosions. In chronic cases the mucous membrane is pale, thickened, wrinkled and covered with thick, ropy mucus. The swollen follicles and mucous gland project above the level of the mucous membrane in the form of nodules, which are about the size of a grain of millet.

In severe cases (*pharyngitis parenchymatosa et purulenta*), the highly congested mucous membrane either becomes œdematously swollen, in consequence of a serous infiltration, and may even present a glassy and gelatinous condition; or it appears as if scalded, and is covered by a purulent, or dirty ichorous, necrotic, or greenish greasy coating, under which losses of substance to a various extent are found, and consequently the mucous membrane frequently presents a somewhat "gnawed" aspect. The lymph follicles and mucous

glands are swollen to the size of peas, or are suppurating. Incisions into the mucous membrane, particularly in the region of the tonsils, often show numerous plugs of pus, which may become partly changed into concretions of lime.

If the affection of the mucous membrane spreads to the submucosa, it will assume a more phlegmonous character (*pharyngitis phlegmonosa*), in which case the submucosa becomes infiltrated with a serous fluid or with hæmorrhages, and is often changed into a gelatinous or purulent substance. In this way, abscesses of various size form under the mucous membrane, lift it up from its base, cause it to become bulged and perforated, or render it gangrenous by the tension they exert on it. The purulent, phlegmonous breaking down of the submucosa may proceed to a wide extent along the loose connective tissue, as, for instance, from the pharynx at the posterior surface of the œsophagus and of the trachea, to the entrance into the chest. This process gives rise to new abscesses and long fistulæ; the mucous membrane often throws off shreds of dead tissue and becomes ichorous; and particles of food, which maintain and increase the suppuration and necrosis, accumulate under the detached mucous membrane. In one case of this kind, Bruchmüller saw the mucous membrane of the entire œsophagus, loosened by the penetration of food.

The retropharyngeal lymph glands, upper cervical glands, and sometimes even the submaxillary and sublingual lymph glands also simultaneously participate in the inflammatory process. These lymph glands in the neighbourhood of the pharynx and larynx get swollen and hæmorrhagically infiltrated, and form abscesses or become disintegrated into a dark red, pulpy mass. The so-called retropharyngeal abscesses are usually full of thick, white, creamy pus, and are either simple or winding. They may contain a fifth of a pint of pus or more. Later on the pus often turns caseous, and becomes encapsuled by a horny hyperplasia of the surrounding connective tissue. In severe cases, the submaxillary and sublingual lymph glands suppurate, and give rise to fistulæ which open outwards.

The mucous membrane of the larynx is frequently congested, diffusely or in spots, and infiltrated in some parts with serous fluid (œdema of the glottis); the epiglottis is œdematously swollen at its base, and the guttural pouch sometimes contains thickened masses of pus. This filling of the guttural pouch does not occur as often as is supposed; for in many cases

sub-parotid and retropharyngeal abscesses are evidently mistaken for it, as we may see when they are surgically opened. The mucous membrane of the Eustachian tube often becomes thickened. The bronchi also suffer from catarrhal changes, sometimes become plugged with pus, and their mucous membrane ulcerated, with the result that the cartilage is laid bare. The lungs often show circumscribed pneumonic foci with necrosis (pneumonia due to foreign bodies), and are filled with stinking ichorous cavities of the size of a pigeon's egg, in the neighbourhood of which pleuritic coatings form. The Schneiderian membrane is usually excessively congested (either brick-red or discoloured), covered with clots of blood or pus, which may assume a croupy appearance, and which at times can only with difficulty be detached. We may also find the under part of the mucous membrane here and there velvety or villous, and the villi provided with blood-filled vascular loops. The oral mucous membrane is often catarrhal, which condition may spread to the intestines. We occasionally meet with a desquamative inflammation of the mucous membrane of the rectum.

In a protracted course of the disease, the *heart* shows a parenchymatous and fatty degeneration; and the *liver* exhibits swelling and fatty degeneration. These symptoms, so far from being peculiar to pharynx-angina, are common to all diseases which are accompanied by high fever and great exhaustion.

With respect to the anatomy of the pharyngeal mucous membrane when in a normal condition, the following points are of importance in the pathology of pharyngitis. The mucous membrane of the anterior surface of the velum palati contains, according to Franck, a thick layer of yellowish, acinose, mixed glands, namely, mucous glands and serous glands (Ellenberger's palatine glands), interspersed among which we observe (only in pigs) numerous small lymph glands. The surface of the pharyngeal cavity possesses, on the contrary, only a very thin layer of these mixed glands. At the base of the tongue near the tonsils we find the so-called follicular glands, which represent the lymph glands of the tongue. The dorsal part of the pharynx contains many lymph follicles, along with numerous mucous glands on its upper edge, and particularly in the neighbourhood of the orifices of the Eustachian tubes. It is noteworthy that in horses and pigs the tonsils, which lie in the posterior part of the oral cavity, possess many *foramina coeca*, whilst in the other domestic animals there is only a single deep fossa, which fact may perhaps partly account for the frequency of angina in horses and swine.

Symptoms in Horses.—1. *Pharyngeal symptoms.* A veterinary surgeon has seldom the chance of observing the

and hot. The swelling is usually bilateral, but, as a rule, is not equally intense on both sides. It is either flat and diffuse, so that the normal outlines become somewhat indistinct, or it is sharply defined; although it may become exceedingly extensive and fill out the entire space between the lower jaw and the atlas. In most cases it is the lower part of the parotid gland which becomes prominently raised by the inflammatory infiltration of the lymph glands that lie under it. In exceptional cases the supporting tissue of the parotid gland may participate in the swelling, and also the guttural pouches. Bell has seen an instance of empyema of the guttural pouches, with temporary deafness, during the course of an attack of pharyngitis in a horse. Also the skin in the neighbourhood of the larynx may become swollen in consequence of collateral œdema, and we may find tumefaction of the glands of the intermaxillary space, submaxillary, sublingual, and sometimes of the thyroid gland. The swellings in the parotid region are generally very painful.

4. The *digestive apparatus* frequently shows symptoms of severe intestinal catarrh; the patient often yawns; peristaltic action is more or less suppressed, and may become entirely arrested; the fæces are stinking, broken up, soft and covered with mucus, and the urine is acid. If there be no intestinal catarrh, and no great diminution of appetite, the urine will retain its alkaline reaction, despite the fact that high fever may have existed for weeks. In severe cases the urine contains albumen. A very low specific gravity of the urine is a striking pathological sign which is sometimes present. Thus we have observed cases in which specific gravity of the copiously discharged urine varied during the three days from 1001 to 1006 (normal s.p. 1014 to 1037); the specific gravity appearing to be dependent on the severity of the disease.

5. *Fever*. The nature and mode of appearance of the fever of pharyngitis is by no means typical. It may be absent in mild cases, or the pulse may show only a slight acceleration, as for instance, up to 48 beats in a minute. In other instances the disease sets in with high fever; the temperature being as high as 104° F., or 105° F., or even higher, while the pulse for the first few days is little, if at all, increased in frequency. We have cases in which, with a temperature of over 104° F., the pulse did not exceed 40 beats a minute during the whole course of the disease. As a rule the pulse gradually follows the temperature, as long as the action of

the heart is influenced by the prolonged effect of such high fever. If the disease begins on the very first day with high fever, we must conclude, as in other diseases, that it has been caused by infection. Pharyngitis, which appears during the course of strangles, is also distinguished by high fever. A gradual rise of temperature to a considerable height points to an intense inflammatory process, especially of a phlegmonous nature. A sudden rise of temperature is usually connected with the formation of abscesses in the lymph glands, etc. A marked rise in the frequency of the pulse is always a bad sign. In the horse, 80 pulsations in a minute are ominous; but a high degree of temperature is not so serious. High fever manifests itself not only by the frequency of the pulse and the temperature of the rectum, but also by unequal distribution of heat in the surface of the body, as for instance, cold ears and cold extremities, with considerable injection of the conjunctiva.

6. Finally, we have observed in the course of pharyngitis a blotchy rash, appearing suddenly and as suddenly disappearing, and occurring a secohd time and disappearing with equal rapidity.

Process.—In the great majority of cases the course of pharyngitis is acute, although certain sequelæ and complications may protract the duration of the disease. True chronic pharyngitis is found only when the action of the exciting irritants is long continued, as in the case of bots or tumours in the pharynx. As a rule, the disease ends in resolution, particularly in cases of simple catarrhal angina, which recovers in from six to fourteen days. On the other hand, cases of pharyngitis in which there is phlegmonous inflammation of the submucosa, or suppurative infiltration of the retropharyngeal and subparotid lymph glands, are of longer duration. The abscesses which form in such cases produce, by the size of the collateral swelling in their neighbourhood, difficulty of breathing and increase of temperature, and may thus protract recovery for four or five weeks, or longer, or may cause death by suffocation, in consequence of a rapidly forming œdema of the glottis. These abscesses rarely open outwards; but, probably more often than is thought, break inwards by necrosis of the pharyngeal mucous membrane, and their contents are then discharged through the nasal and oral cavities, when the dyspnœa, as a rule, will be suddenly relieved; or they may run into the larynx and trachea, in which case the patient may be suffocated. Sometimes a necrotic inflammation of the

guttural pouches, hyoid bone, and even of the occipito-atloid articulation may become developed (Ridge). In one case, a secondary ulcer of the guttural pouches of about the size of a shilling gave rise to a hæmorrhage which terminated fatally (Eder). In very rare cases an abscess opens both inward and outwardly, and thus produces a pharyngeal fistula, as Romant observed in a foal, out of which fistula milk escaped while the foal was suckling. We have seen two similar cases in horses. In one of these, after the abscess, which was the size of a man's fist and at a depth of four inches in the region of the parotid gland, had been surgically opened, it burst spontaneously inwards. In the other case there remained after recovery a running fistula, through which fluid could be injected into the pharynx and mouth. Parts affected by burrowing and perforating suppuration may become ichorous, which is a condition of great danger, as it may lead to septic poisoning, when the temperature is extremely high, the breath has an intensely putrid smell, and the animal coughs up necrotic, fœtid shreds of tissue.

If the inflammatory infiltration of the glands does not lead to the formation of abscesses, and to the outward discharge of their contents, the glands are liable to become indurated, caseous and calcified, which condition causes a permanent increase in the circumference of the region of the larynx and pharynx, and thereby produces more or less difficulty in breathing. Paralysis of the pharynx has been observed as a sequel of pharyngitis.

Inflammation of the lungs is a dangerous, and by no means rare, complication of phlegmonous and necrotic pharyngitis, and is the most frequent cause of death. As in severe cases the possibility of this complication is always present, it is the imperative duty of the veterinary surgeon carefully to examine the chest during the course of the disease. This inflammation of the lungs is in most cases due to foreign bodies, and is caused by portions of the oral contents having passed down the trachea. It is characterised by rapid gangrenous destruction of the affected parts of the lungs, and may be produced by forcible or unskilful drenching, or by emboli, which arise from the ichorous foci of the necrotic pharyngitis (metastatic pneumonia).

Differential Diagnosis.—It is easy to diagnose a developed and uncomplicated case of pharyngitis in the horse, but is often hard to distinguish it in its early and later stages

from other affections which have similar symptoms. Thus, confusion may occur in cases of foreign bodies being lodged in the mouth, pharynx and œsophagus, and with new growths in the pharynx, particularly polypi, actinomycomata and sarcomata, which render deglutition difficult. Affections of the pharynx, such as stenosis, paralysis and spasms of the part, often have similar symptoms; and parotitis, glossitis, stomatitis, swelling of the guttural pouches, ptyalism, etc., may lead to confusion. At the beginning of this chapter we have alluded to strangles, which of all complaints can be most easily mistaken for pharyngitis.

Prognosis.—Prognosis depends on the intensity and extent of the affection and on the complications that are present. Although a mild attack of catarrhal pharyngitis quickly recovers, the prognosis of a somewhat severe one should always be made with reservation, because sudden and unexpected death may ensue from gangrene of the lungs, or œdema of the glottis. Much also depends on whether or not the inflammation has spread to the larynx.

Therapeutics.—In an enzootic outbreak the affected horses should be segregated as a prophylactic measure. The stable should be of medium temperature, well ventilated, but free from draughts, and the food easy of mastication and deglutition. It is consequently advisable to give, if practicable, only green food, or very soft hay, or perhaps mashes of meal and bran. Oats and chop should be entirely avoided. Care should be taken that fresh water is kept constantly before the horse. The lips and edges of the nostrils, which get covered with mucus and crusts should be carefully cleaned, and the mouth frequently washed out with cold water or with some mouth douche, such as solutions of salt and vinegar, potassium chlorate, etc. If the mouth be rinsed out with a syringe, this instrument should be used very carefully, and the jet be directed towards the cheeks, and not in the direction of the pharynx. The surroundings of the patient should also be kept scrupulously clean.

In severe phlegmonous processes accompanied by great pain, tension and heat, it is advisable in the beginning of the attack to put cold applications or even ice round the throat; and later on, when the abscesses begin to form, or even when the swellings are of a less inflammatory character, Priessnitz's bandages are of the greatest use, especially if the inner layers of the

bandage are moistened with carbolised water instead of with ordinary water. These bandages hasten the ripening of the abscesses and soften the hard inflammatory swellings.

The inunction of grey mercurial ointment on the swollen parotid region is much practised ; but it is doubtful whether sinapisms and stimulating ointments applied to the region of the parotid gland and intermaxillary space are of any avail. They certainly should not be used indiscriminately. All that can be said for them is, that they hasten the maturing of the abscess more than moist heat. There is no ground for the idea that they have a derivative effect from without. They might be used with advantage on very circumscribed hard swellings, which require to be quickly softened. On the other hand, these severe inunctions should not be employed in cases of widely extended swellings, severe local inflammation, deep and diffuse suppuration, impaired action of the heart, or general debility, nor with languid young animals of weak constitution, because their application may give rise to enormous swelling, considerable dyspnœa and rapid gangrenous destruction of the cellular tissue, in consequence of which many horses have died.

After we have ascertained by palpation the presence of an abscess, we should lose no time with blistering ointments, but should open it as early as practicable by cutting the skin, and then penetrating downwards with the fingers in a preparatory manner. We may thus open abscesses at a depth of four inches. Their further treatment is a matter of surgery.

Inhalations of steam, either plain or medicated with carbolic acid or creolin are much used ; but we should avoid having them too hot. Moreover, their value is probably insignificant. The local employment of astringents is of more importance, although it cannot be easily practised on horses. Kagel advises the injection of medicines into the pharynx by introducing an india-rubber tube into one of the nostrils. The tube used for this purpose is about two feet long, has a thick wall, and small calibre. About 3 oz. of the fluid is injected, and consists of ½ per cent. solution of common salt, to $\frac{1}{10}$ per cent. of corrosive sublimate, etc. The astringent or disinfecting solutions (½ per cent. of nitrate of silver, Lugol's solution, 1 per cent. of lactic acid, ½ to 1 per cent. of alum) recommended by Dieckerhoff for intrapharyngeal injections, sometimes render good service ; but are not always without danger, as we can state from our own experience.

We should be cautious in administering drugs internally,

and under all circumstances fluid medicines should be avoided. Electuaries and balls should be used only to a small extent. Fever and constipation are the sole conditions that require internal medicines, and we should try to combat them by frequent enemas of solutions of common salt and of soap and water, and by subcutaneous injections of appropriate remedies, as for instance, physostigmine for constipation, if dyspnœa be not present, or a solution of tartar emetic, for preference, in the drinking water. Pilocarpine and arecoline, are contra-indicated in pharyngitis, because these aperients produce at the same time copious salivation, which may become dangerous if the power of swallowing be impaired. The high fever of pharyngitis often resists the action of febrifuges, and we have frequently to fall back upon cold enemas and cold packing of the trunk. Antipyrin is the best febrifuge, because it can be used subcutaneously. It is injected in doses of from 150 to 300 grains dissolved in equal parts of water. In septic fever subcutaneous injections of spirits of camphor (150 to 300 grains, hourly) are indicated.

It may be necessary to perform tracheotomy in cases of pneumonia following on pharynx-angina, or hyovertebrotomy in order to empty distended guttural pouches. As success from tracheotomy depends upon its being performed at the proper moment (we cannot expect a real benefit from it, if we wait too long), and as the operation may become necessary at any moment, the instruments for its performance must be kept in readiness. Pneumonia due to foreign bodies, or metastatic pneumonia is to be treated according to general principles. The treatment, however, is often unsuccessful.

Pharyngitis of other Domestic Animals.—There is so great uniformity in the chief symptoms of pharyngitis, that a detailed description of the disease in our various domestic animals is superfluous. With regard to cattle, we may refer to the morbid conditions in horses. The chapter on actinomycosis may be consulted in the treatment of this disease. In the following remarks we shall discuss pharyngitis, particularly in pigs and dogs :—

1. Pharyngitis of pigs, which frequently occurs, has been fully described by Spinola in his "Diseases of Pigs." In swine, as in other animals, it is easily confused with larynx-angina. We must also distinguish between pure idiopathic angina and anthrax-angina. Swelling of the throat is one of the most prominent symptoms in anthrax in swine. According to Spinola, the principal cause of pharyngitis is the drinking of very cold water when the body is heated. On which account the disease is particularly prevalent during spring in mountainous districts among pigs which graze near mountain streams. We have also to

consider mechanical and chemical causes. As an instance of the former: In Indiana, in 1900, a number of swine died from traumatic pharyngitis after feeding on spelt wheat and barley stubbles. The beards of the grain lodged in the mucous membrane of the throat, causing such pronounced swelling that the animals died of asphyxia.

The symptoms of pharyngitis in swine vary according to the severity of the disease. In slight cases there is little or no fever. We chiefly find congestion of the visible mucous membranes, frequently lachrymation, accelerated and more or less difficult breathing, dry cough, hoarseness, slightly increased temperature of the mouth, difficult deglutition, retching, vomiting and swelling and congestion of the pharynx, which is hot and painful. Although the appetite is impaired, it is seldom suppressed. The disease runs its course in from ten to fourteen days, when the cough gradually becomes moist, and recovery as a rule takes place. According to Spinola, the symptoms of severe inflammation are: considerable congestion and dryness of the snout and visible mucous membranes, especially that of the mouth; shining eyes; staring look; the region of the pharynx is congested, abnormally warm, swollen and painful; high temperature of the mouth; oppressed and accelerated breathing, which in severe cases is very difficult; indistinct, short and painful cough, which the patient tries to suppress, when the breathing is impeded; hoarse grunting; diminished or lost appetite; difficult deglutition; retardation of defecation and urination; hard condition of the fæces; and yellow-brown (fiery) colour of the urine.

These symptoms naturally increase in intensity when the disease assumes a more severe type. The mucous membrane becomes deeply congested and livid; respiration difficult and almost spasmodic; the patient sits on his hind quarters with his head stretched forward and his fore legs wide apart, in order to get air into the lungs through the open mouth, from which panting and whistling sounds issue; there is great anxiety and uneasiness; the eyes are protruded and wildly staring; the veins of the head are swollen; the snout, lips and intermaxillary space down to the throat assume a purple colour; and the patient begins to stagger, falls down, and, becoming convulsed, dies from suffocation after the disease has run a course of from twenty-four hours to three days. If the disease does not terminate fatally, recovery may take place in from five to ten days with gradual decrease of the symptoms, in which case hard painless swellings frequently remain in the intermaxillary space, the presence of which often more or less obstructs the entrance of air into the larynx and imparts a panting character to the breathing. According to Röll, these swellings are the remains of small, empty, circumscribed abscesses, surrounded by infiltrated connective tissue.

In addition to pronounced symptoms of inflammation of the pharynx and larynx, *post-mortem* examinations frequently reveal croupous deposits in the larynx and upper part of the trachæa (Sesselmann), the so-called membranous croup. Symptoms of asphyxia may also occur, as extreme œdema, etc., of the lungs, hyperæmia with its consequence to the blood-vessels of the brain.

Prognosis depends on the causes and the possibility of removing them, on the degree, and particularly, on the extent of the affection, and on timely application of appropriate treatment. The more the larynx is implicated, and the more dyspnœa present, the more unfavourable will

be the prognosis; because suffocation is the most frequent and rapid cause of death. The degree of fever has also to be taken into account.

As regards *treatment*, it is necessary, above all things, to provide an abode which is dry, moderately warm, and free from draughts; and food consisting of meal and bran mashes, green fodder, or sour milk. At the beginning of the disease it is well to give an emetic, which sometimes does not act, and can be repeated next day. For this purpose white hellebore (8 to 30 grains) is the most appropriate agent, and should, if practicable, be given in the food. Ipecacuanha (15 to 45 grains) may be used instead. When the animal will not eat, the emetic may be mixed with fat. Drenches should not be employed; for which reason tartar emetic is not very appropriate. For these cases we can recommend the rectal application or subcutaneous injection of veratrin (.3 to .5 grains in a drachm of spirit). Apomorphine has no effect on pigs. If there is much difficulty in swallowing, we should abstain entirely from giving medicine by the mouth, for so doing would be almost certain to cause pneumonia. Occasionally the veterinary surgeon is called in too late for treatment, in which case the animal should be slaughtered as quickly as practicable, unless circumstances (pregnancy, state of nutrition, breeding value, etc.) suggest the expediency of giving it a chance of living. Stimulating inunctions (Ungt. Cantharid., etc.) on the region of the larynx have sometimes good results. Priessnitz's bandages are worthy of recommendation. Local treatment of pharyngitis is not usually practicable with pigs. Lövy states that he has seen good results from painting the pharyngeal mucous membrane with Lugol's solution. Meyer recommends energetic massage of the throat with rags saturated in paraffin oil.

2. *Pharyngitis of dogs* is also a not uncommon affection, and is one in which relapses are numerous. According to the statistics of the Berlin canine hospital, the frequency of pharyngitis in dogs amounts to one-third per cent. of all cases of disease. The finer breeds, such as pugs, terriers and greyhounds are most susceptible to it. The disease begins with impairment of prehension, difficulty of swallowing, and salivation. A painful swelling of the sub-parotid lymph glands, which in very rare cases suppurate, quickly makes its appearance. On inspection of the pharynx we can generally see severe congestion and swelling of the mucous membrane of the pharynx, tonsils, soft palate, etc. The course is usually benign, although the disease generally lasts for a considerable time (14 days to 4 weeks). Sometimes deafness occurs, apparently in consequence of the spreading of the inflammation of the pharynx through the Eustachian tube to the middle ear. The treatment of pharyngitis with little or no fever, consists chiefly in the application round the throat of Priessnitz's bandages, which should be removed every three hours. The swollen glands may also be rubbed with camphor liniment, etc. A local treatment of the affected pharyngeal mucous membrane can be practised by painting it with astringent fluids, such as lime water, and solutions of nitrate of silver (½ to 1 per cent.), tannin, alum (1 to 2 per cent.), etc.

In cats, traumatic pharyngitis is the form most frequently met with (Nadeln). Gray observed diphtheritic pharyngitis in three Persian cats with diphtheritic membranes in the pharynx and larynx. Diphtheria bacilli, however, were not traceable.

CHAPTER II.

DISEASES OF THE ŒSOPHAGUS.

Classification.—The occurrence of diseases of the œsophagus are as rare as their diagnosis is difficult. Some pathological processes, as, for instance, those caused by foreign bodies, belong more to surgery than to medicine. We, therefore, propose to discuss under this heading only the following affections of the œsophagus :—

1. Dilatations.
2. Constrictions.
3. Lacerations.
4. Paralysis.
5. Spasms.
6. Inflammations.

DILATATIONS OF THE ŒSOPHAGUS.

Etiology.—The dilatations of the œsophagus may be divided into diffuse expansions which involve the entire periphery of the œsophagus, and into circumscribed ones of a certain part of the wall (œsophageal diverticula). The ectasis usually occurs along with stenosis of the œsophagus, and in that case always above it. The ectasis develops in a manner similar to that of a dilation of a ventricle of the heart with stenosis of the valves. The muscles of the œsophagus become gradually relaxed, owing to over exertion in trying to press accumulated masses of food through the narrowed part of the œsophagus, which food accumulates above the constriction, dilates the walls and causes the muscular coat to become atrophied and pale. Ectasis without stenosis is rare, and seems to depend on local inflammatory changes of the walls, or on injuries, as in the case of sudden expansions of the mucous membrane hrough the split separated muscular coat. Possibly the presence of Me'scher's tubes (psorospermiæ)—often found in the œsophageal muscular apparatus of the horse—plays a certain part in bringing about these dilatations.

Diverticulæ are divided according to their mode of development into diverticulæ by pressure and diverticulæ by traction. The former are produced by the pressure from within of a ball of food on a part of the wall which is more yielding than the remainder, as a result, perhaps, of previous injury to the wall. The latter are usually the consequence of a cicatricial contraction of the peri-œsophageal tissue, or of a neighbouring organ, as, for instance, glands, in a manner analogous to the formation of bronchiectasis in cases of interstitial pneumonia.

The dilatations are either defined, in which case they are usually spindle-shaped, or they implicate the entire œsophagus. Stockfleth once found the œsophagus in a horse dilated to a length of 54 inches, with a diameter in the middle of 3½ inches. Diverticulæ may hold as much as a bucket, and may become adherent to neighbouring parts, as, for instance, to the trachea. Dilatations and diverticulæ of the œsophagus are not very rare in the domestic animals, and in horses and cattle are generally met with in the cervical portion of the œsophagus. They are also found in sheep, goats and dogs.

According to Storch some horses have congenital diverticula, or at least a predisposition to contract them, the second pharyngeal cleft probably participating in this formation. He also considers that the imperfect internal pharyngeal fistulæ are possibly connected with the pouch-like diverticula of the œsophagus.

Symptoms.—As these dilatations become developed very slowly, it is difficult at first to recognise their presence. The earliest marked symptom is a capricious appetite, which is manifested by excessive hunger on some occasions, and by rapidly produced satiety on others. Gradual emaciation sets in, and the rate of feeding often becomes very slow. This condition may last for years without its cause being recognised. In most cases it is accompanied by vomiting, which appears sooner or later after feeding, and recurs frequently, periodically and with a regular duration. Œsophageal regurgitation is sometimes effected through the nose, and is occasionally accompanied by a painful cough. The thrown-up masses are usually covered with frothy mucus, and if they have remained some time in the œsophagus, may be more or less decomposed; but they will in no case be digested, as has often been erroneously stated. This mistake has probably arisen from the fact that food may be brought up from the stomach at the same time. Independently of these

discharges of food, there is frequently a flow of much mucus mixed with food, from the mouth and nose. Usually deglutition is very difficult or impossible. The animal sometimes tries with the greatest exertion to swallow a mouthful, while keeping his head and neck considerably flexed, and the hind legs placed under the body, in which case we often hear peculiar gurgling sounds. It occasionally happens that the animal, without having eaten, goes through the movements of mastication and deglutition, and at the same time slavers a good deal, and has frequent colicky pains, which often come on periodically. In cows tympanites, suppressed rumination, and, as some state, pica (p. 42) have been seen. In certain cases the animal can readily swallow oats and chop, but not hay.

One of the most important symptoms of dilatation of the cervical portion of the œsophagus is the appearance, on the left side of the throat immediately after eating, of a swelling which can be emptied by pressure, and which may attain the size of a man's head, and may also be immovable. We may also find on the left side of the throat a longish tumefaction of the œsophagus, which, when œsophagitis supervenes, becomes painful to the touch, and exhibits very marked peristaltic movements during deglutition. On the introduction of a probang, we find an obstacle at the site of the stenosis, which is situated below the dilatation, or at the pouch-like diverticulum, or where the masses of food have accumulated. In rare cases the swelling occurs on the right side of the throat.

These symptoms of the digestive apparatus are very often complicated with cough, dyspnœa and attacks of suffocation, and even fatal asphyxia may arise from compression of the trachea by the enlarged œsophagus. In other cases fatal pneumonia, due to defects of swallowing, may also occur. In one instance strong pulsations were found in the left carotid artery. The disease, which usually lasts for years, produces gradual debility and emaciation, and may end fatally by suffocation, by pneumonia due to foreign bodies, or by rupture of the greatly distended œsophagus. Septicæmia sometimes appears to become developed from ulcerous changes in the œsophageal mucous membrane.

Prognosis.—Prognosis is nearly always unfavourable. This disease may easily be mistaken for rupture, inflammation, spasm, or paralysis of the œsophagus; abscesses in parts adjoining the œsophagus; pharyngitis; stomatitis; acute

gastric catarrh; poisoning and colic. The same remark applies to the other affections of the œsophagus. We can, however, make without difficulty a correct diagnosis by a careful examination of the œsophagus with a probang, and particularly by paying attention to the long continuance of the disease.

Therapeutics.—As treatment is not very successful, we have to rely chiefly on a fluid or semi-fluid diet, and on frequent feeding with small quantities at a time; and we may try, by means of the probang and by massage, to prevent occlusion by food. Little is known about the operative treatment of a diverticulum. Stockfleth, following the example of Moisant and Colin, recommends extirpation of the dilated part of the wall, and union, by suture, of the edges of the wound. Schindelka cured a case in a horse by these means. Tracheotomy instruments should be kept in readiness, on account of severe dyspnœa being likely to supervene.

CONSTRICTIONS OF THE ŒSOPHAGUS.

Etiology.—Stenosis of the œsophagus may occur in various ways. The congenital form is the rarest. It may be caused by compression of the œsophagus from without, as in the case of tumours, such as strumata, actinomycomata, lymphomata, periœsophageal abscesses, especially in cattle; abscesses and tubercular degeneration in the bronchial and mediastinal glands; aneurisms of the aorta and melanomata. These tumours may exert pressure either laterally or in the form of a ring. Stenosis due to narrowing of the lumen of the œsophagus from within (obturation-stenosis) is usually caused by impacted foreign bodies (bones, potatoes, pieces of carrot, pieces of bread, cartilage, apples, physic-balls, pieces of food, broken-off tracheotomy tubes, etc.). It may also be produced by tumours which project into the lumen, as, for instance, papillomata, carcinomata and actinomycomata in cattle; by tumours which contain spiroptera sanguinolenta, and by inflammatory hypertrophy of the muscles of the œsophagus in dogs. Also strictures may be set up by cicatricial contraction of the mucous membrane in consequence of erosions or injuries, or by a cancerous degeneration of the walls. They may take the form of a thick muff with a rough surface, or of a ring; or the thickened cicatricial tissue may project into the lumen in the shape of a valve. We shall discuss later on (p. 37) the stenoses

which are caused by contraction of the œsophageal muscles. Particulars about foreign bodies in the œsophagus will be found in text-books on surgery.

Symptoms.—As the above-mentioned forms of stenosis are frequently complicated, later on, with dilatation, the clinical aspect of stenosis on the whole differs but little from that of œsophageal ectasis, the symptoms of which may be referred to here. Chronic tympanites is frequently a very characteristic symptom of œsophageal stenosis in cattle. As stenosis of the œsophagus in cattle is usually caused by the compression of a tuberculous bronchial or mediastinal gland, we can diagnose this form when other and less well-marked symptoms of tuberculosis (cough, emaciation, dulness on percussion, friction sounds, etc.) are accompanied by chronic tympanites, without any other digestive disturbances being present. (See chapter on Tuberculosis, Vol. II.). In dogs and cats, the presence of metallic foreign bodies in the œsophagus may be detected by means of the X-rays. By this means, Porcher and Morey were able to discover a teaspoon in the œsophagus of a dog.

Therapeutics.—The treatment of stenosis, and especially that of cattle, produced by compression and accompanied by tympanites, belongs to the province of surgery, and consists in the frequent use of the probang. We might, in a manner similar to the treatment of stenosis in man, try to widen the constricted part by the introduction of probangs of successively increasing thickness. The dietetic principles are the same as in dilatation of the œsophagus. In the case of foreign bodies in the œsophagus, before resorting to an operation it is advisable to try what arecoline or pilocarpine will do with horses; while dogs and swine should be given an emetic, such as apomorphine or veratrine administered sub-cutaneously.

LACERATIONS OR PERFORATIONS OF THE ŒSOPHAGUS.

Etiology and Symptoms.—Tearing of the œsophagus from without inwards arises from external injuries, especially kicks by horses and falls. Perforations from within outwards may be due to impacted foreign bodies, careless introduction of a probang, and bursting of the very thin wall of a diverticulum, or muscular wall which has suffered from fatty degeneration, even if deglutition be normal. The symptoms vary according

as the œsophagus is torn in its cervical part or in its thoracic part.

Rupture of the cervical portion of the œsophagus manifests itself, first of all, by a swelling which is often enormous, and which is caused by the escape of fodder, and is complicated later on with a phlegmonous inflammation. Swallowing being impossible, regurgitation of food and drink takes place through the mouth and nose. In some cases the entrance of air into the connective tissue which surrounds the œsophagus causes a diffuse emphysematous swelling of the throat, head and shoulders. The formation of an œsophageal fistula, and even of an œsophago-tracheal fistula has been observed, and in these cases the expired air has a putrid, gangrenous smell.

The cases of rupture of the thoracic portion of the œsophagus are still more severe. The animal exhibits sudden vertigo, and falls down during work, or presents to the spectator symptoms of violent congestion of the lungs and dyspnœa, and sometimes apparently those of colic. Pneumo-thorax is an occasional complication. In these cases the patient shows great anxiety, trembles, evinces a desire to vomit, and stands with the head stretched straight out, while the muscles of the neck are violently contracted. Symptoms of purulent ichorous pleuritis gradually appear, with the result that the animal dies. In one case, that of a dog, a foreign body, after perforating the wall of the œsophagus, passed into the aorta, causing hæmorrhage into the œsophagus, stomach and bowels. Hæmatemesis of a fatal character supervened (Almy).

Therapeutics.—Ruptures of the thoracic part of the œsophagus are not amenable to treatment; but an animal suffering from a rent of the cervical portion of the gullet may sometimes be saved by prompt operative treatment, which should consist of free incisions into the tumour and reuniting the adjusted edges of the wound by sutures. (See Sander's case in *The Veterinarian*, 1851.)

PARALYSIS OF THE ŒSOPHAGUS OR PHARYNX (*Dysphagia paralytica.*)

Etiology.—Paralysis of the œsophagus and pharynx often occur together, and consequently cannot be distinguished from each other in practice. It originates either centrally, in the brain, or peripherally, in local changes of the œsophagus and pharynx. The central causes are paralysis of the centre

of deglutition in the brain in consequence of hæmorrhage, concussion of the brain, abscesses, new growths, parasites, and particularly inflammation of the brain (subacute inflammation of the brain in horses). Paralysis of the centre of deglutition is observed in the course of some poisonings, such as pathogenic fungi (tilletia caries, and mould fungi), in herbivora, and ptomaine poisoning (sausage poisoning) in carnivora. Uterine paralysis and rabies are infective diseases which set up cerebral paralysis of deglutition. The local causes consist in inflammation of the pharynx and œsophagus, due to the inflammation spreading from the mucous to the muscular coats. These inflammations are either primary or secondary, as when they occur during the course of strangles or petechial fever.

Dexler is of the opinion that paralysis of the œsophagus in the horse is frequently due to a combined affection of the 9th-12th cranial nerves. He considers, however, that it is only very rarely that such a diagnosis would be justified by the clinical aspects of the case. He also considers that a clinical distinction between peripheral and central paralysis is, as a rule, impossible.

Symptoms.—The principal symptom of paralysis of the œsophagus and pharynx is inability to swallow, in consequence of which saliva and masticated food dribble continuously from the mouth. When the œsophagus alone is paralysed, it is sometimes filled with food from the pharynx down to the stomach, which condition is manifested externally by a cylindrical swelling on the left side of the throat. The symptoms in other respects are similar to those of ordinary affections of the œsophagus. Fatal pneumonia, due to foreign bodies, appears occasionally at a later stage of the disease.

Paralysis of the organs of deglutition is generally complete or bilateral; but in exceptional cases we may find a unilateral paralysis of deglutition, when the originating paralysis is restricted to one side of the pharynx. Schöttler describes a case of periodic œsophageal paralysis in a mare.

Therapeutics.—The treatment of paralysis of deglutition is confined within narrow limits. Besides the application of external irritants, such as blistering ointments, to the region of the pharynx and œsophagus, and the introduction of the probang, we may try internal stimulants in the form of subcutaneous injections, as, for instance, strychnine and veratrine. Electricity

may also be employed. Sometimes a spontaneous recovery takes place.

We must not confuse inability to drink water with paralysis of deglutition, because otherwise healthy animals, which are able to masticate and swallow dry food in a normal manner, cannot suck up water, the functional failure being generally due to congenital causes, owing probably to some defect in the organ of suction. The affection appears not only in sucklings, but also in full grown animals, particularly horses. Straub has described such a case, and we have treated one in an eight-year-old horse, which was brought to us with the report that "it drank like a dog." This animal, which was otherwise quite healthy and able to eat dry food in an ordinary manner, could not perform sucking movements. Although it played with the water and tried to drink, it was quite incapable of doing so. The only treatment was to give the animal as much water as possible with its food.

SPASM OF THE ŒSOPHAGUS OR PHARYNX (*Œsophagismus*).

Independent spasmodic stenoses (*dysphagia spastica*), as compared to symptomatic spasmodic contractions of the œsophagus, with which they are often confused, occur but rarely. Spasmodic stenosis in horses, identical in character with the purely nervous condition known as œsophagismus, is frequently provoked by morphia-injections, chloroform or chloralhydrate anæsthesia (Froehner and Vennerholm); a morsel of food in the gullet; an ulcer upon, or other injury to, the wall of the œsophagus. In cats, the symptoms frequently manifest themselves after the swallowing of a pin. Mollereau has observed them in a very nervous Percheron; Guilmont, in a horse; and Mossé, in a hinny and in foal mule. The spasmodic attacks in these cases lasted from three-quarters of an hour to six hours. In the case of the Percheron, the animal was convulsed, vomited, placed its feet under its body, and stretched out its head. In the Guilmont case, the foal suddenly began to paw the ground and showed other signs of colic, could not swallow, and made attempts to vomit when one pressed on the tense œsophagus. The treatment consisted in the administration of bromide of potassium. Mossé effected a cure by œsophagotomy, and by the insertion of an œsophageal tube. Friedberger has observed an instance of periodic spasmodic stenosis in a horse. Cadéac has described a very

interesting case of chronic spasm of the œsophagus in a well-bred horse, which, during a year and a half, had several attacks each week. While feeding, he would show great uneasiness, paw the ground with the fore-legs, stretch out the head, discharge frothy saliva from the mouth, and, when trying to vomit, utter shrill or sharp hissing sounds, while at the same time, saliva was ejected through the nose. During the attack the bolus of food could be seen moving violently up and down the œsophagus, and the upper lip was spirally contracted. The vomited food was on no occasion sour. The attacks lasted one to two hours. The consumption of dry, short fodder and of cold water was shown to be the exciting causes of these seizures, for they were experimentally produced with certainty by giving such aliment. Roy describes a similar case, that of an exceptionally gentle-tempered mare, which had five attacks in five years, and, upon each occasion the symptoms appeared after drinking to repletion, and the seizure always lasted five hours.

Spasms of the swallowing muscles occur in rabies, epilepsy, catalepsy and tetanus, and require a casual rather than a systematic treatment. They arise chiefly from contraction of the muscles of the œsophagus, and perhaps of the muscles of the upper part of the gullet. The cases described as "intermittent paralysis of the œsophagus" are no doubt periodic œsophagismus.

INFLAMMATION OF THE ŒSOPHAGUS (*Œsophagitis*).

Varieties.—Inflammation of the gullet may involve the mucous membrane, submucosa, muscles and periœsophageal connective tissue, so that, strictly speaking, we have to recognise a catarrhal, phlegmonous and parenchymatous œsophagitis, and a peri-œsophagitis. The three last-named varieties are usually due only to foreign bodies or wounds.

Causes.—There are various causes of inflammation of the mucous membrane of the gullet. Thus, we find catarrhal œsophagitis occasionally in cases of pharyngitis, and also as a symptom of general diseases, such as rinderpest, foot-and-mouth disease, and sheep-pox. It may be caused by new growths in the œsophagus, or by parasites, especially by *spiroptera sanguinolenta* in dogs. In other cases foreign bodies and especially corrosive substances, such as mineral acids and

alkalis, set up catarrhal and even croupy inflammation of the mucous membrane. For instance, croupy inflammation has been observed in cattle after drenching with ammonia for tympanites; and ulcerous changes have been seen in horses after the administration of undissolved tartar emetic. Thermal irritants may also excite inflammation.

Anatomical Conditions.—The anatomical changes vary according to the intensity of the disease. In a catarrhal and somewhat superficial inflammation, we may find no increase of mucus, as in other inflammations of the mucous membrane, but only increased hyperæmia and considerable proliferation of epithelium. Chronic catarrhs are distinguished by their tendency to papillary proliferation. This peculiarity is explained by the fact that the œsophageal mucous membrane, especially in cattle, closely resembles skin in structure, a fact which Schütz has demonstrated by careful study. It possesses an epidermis, a rete malpighii, and papillæ supported by a layer of connective tissue. Therefore its pathological changes resemble those of the skin, especially as regards desquamation of epithelium and papillary hyperplasia in chronic catarrh. It is to be regretted that in most *post mortem* examinations too little attention is paid to the condition of the œsophagus, and for this reason our knowledge of the anatomical changes in this organ are very incomplete. Kitt found in horses and dogs losses of substance in the form of "gnawed-out spots," a dull grey, greasy epithelial covering, congestion in streaks, petechial hæmorrhages and swelling of the submucosa.

Symptoms.—The symptoms in mild catarrhs are not well marked. In the beginning of an attack it is hardly possible to make a correct diagnosis; but the appearance of difficulty of breathing, attempts to vomit, and increased sensibility of the gullet to pressure from outside, furnishes us with valuable diagnostic data. In a case of croupy inflammation of the œsophagus, which was caused by the corrosive action of ammonia, severe groaning, slavering, coughing and inability to swallow were observed. Also a croupy tube, 15 inches long, and having a thickness equal to the calibre of the œsophagus, was expectorated after a few days.

Therapeutics.—In those cases which can be diagnosed the treatment should be restricted to the application of cold in the form of small pieces of ice for the patient to swallow,

non-irritating watery diet, and the internal administration of astringent solutions (tannin, alum, potassium chlorate, etc.), if they do not upset the digestion. Counter-irritation on the throat is useless. The periodic introduction of a probang may be used to prevent œsophageal stenosis, which might be caused by the cicatrisation of croupy ulcers.

DISEASES OF THE CROP IN BIRDS.

CATARRH AND INFLAMMATION OF THE MUCOUS MEMBRANE OF THE CROP.

Etiology.—Catarrh of the mucous membrane of the crop becomes developed by abnormal retention, drying, and decomposition of food in the crop, by the consumption of putrid food, by overloading, by accumulation of large masses of indigestible materials (such as sand and stones), and by the invasion of parasites, as, for instance, Spiroptera nasuta (Filaria nasuta), according to Robin, and Filaria uncinata, according to Zürn. Intense inflammation of the mucous membrane of the crop is found in cases of poisoning, as for instance, with phosphorus, arsenic, mercurial preparations, common salt, etc.

Symptoms.—In the not uncommon simple catarrh of the mucous membrane (so-called "soft crop"), we find a soft and sometimes a tympanitic swelling of the crop, on account of the consumption of food having almost entirely ceased. The bird tries more or less successfully to vomit, and eructates fœtid gases, the discharge of which can be easily effected by pressure on the crop. The bird is very weak, and often dies from inability to eat.

Therapeutics.—Treatment consists of massage, with the object of pressing out of the mouth the contents of the swollen crop, while the head of the bird is kept hanging down. We may give with a teaspoon astringent solutions of alum, tannin, or sulphate of iron, or a solution of salicylic acid, and, after keeping the bird fasting for one day, we may, for the next few days, give soft food with only a few grains of corn.

OBSTRUCTION OF THE CROP (*Ingluvial Indigestion*).

Etiology.—The obstruction or "hardening" of the crop is usually caused by the consumption of substances which are

more or less indigestible, such as hard, dry corn, and foreign bodies of an extremely varied nature, as, for instance, the leaves of *Robinia pseud-acacia*. Pressure on the œsophagus, produced by inflammation in the adjoining structures, such as the lungs, pleuræ and trachea, may, in exceptional cases, cause overloading of the crop. Railliet and Lucet have described a case of ingluvial indigestion in ducks, which was set up by *trichosoma contortum*. These thread-like worms had settled under the mucous membrane of the crop, and caused the walls to become thin and hyperæmic.

Symptoms.—The most striking symptom is the extraordinary distension of the crop. Zürn saw a hen's crop which, with its contents (food, sand, small stones, etc.), weighed 2 lbs. 14 oz. A hen's crop was in one case found to have a circumference of 16 inches, and a diameter of 6 inches. In these cases, contrary to the condition of "soft crop" above, it is hard and firm to the touch, no food is eaten, and a discoloured, fœtid fluid flows from the beak as a consequence of the catarrh which usually supervenes. Occasionally the tightly stretched wall of the crop becomes ruptured, or a so-called "pendulous crop" is developed, as the result of repeated dilatations of its wall.

Therapeutics.—The treatment, as in that of "soft crop," consists of massage in the form of rubbing, stroking and kneading. Some recommend the administration of hydrochloric acid, a 1 per cent. solution of which we generally use (a tea to a tablespoonful every three hours). Zürn gives one or two drops of this acid in a teaspoonful of an infusion of peppermint or sweet flag (*Acorus calamus*), three or four times daily. As a last means, we may surgically open the crop and remove the accumulated matter. This operation, which should be carried out according to the principles of œsophagotomy, is not applicable to pigeons, because it has been proved that, with the exception of a case reported by de Does, these birds bear it very badly. Zürn ascribes this susceptibility in pigeons to the peculiar condition of the membrane lining the culs-de-sac of the crop (accessory pouches), which, particularly during pairing-time, are very vascular.

VITIATED TASTE

BOVINE PICA (*Licking Disease in Cattle*).

General Remarks.—Among the many pathological aberrations of taste in the domestic animals, the licking disease of cattle and the wool-eating of sheep occupy the first position, both from their scientific interest and from their importance in agriculture. Hence we have deemed it proper to deal with them in separate sections and, naturally, under the diseases of digestion. Other forms of morbid appetite, as in rabies, diabetes insipidus, etc., are merely symptoms, like the aberrations of taste which occasionally occur in mankind during pregnancy, chlorosis and hysteria, and do not demand separate discussion. The same remark applies to many other vitiated tastes which may be regarded as forms of play or vice, as, for instance, eating the after-births.

Pica has no uniform etiological basis, and should rather be ascribed to several primary causes. Thus it follows that it constitutes no specific disorder, and, like colic, is only the most pronounced symptom of certain morbid conditions. Accordingly, it has been the subject of theories more numerous and more divergent than any other disease. It has been variously attributed—at one time to infection, at another to heredity and imitation; and then to a nervous affection; to osteomalacia; to insanitary stabling; and finally to abnormal conditions of food or soil.

According to Roloff and Röll, pica is not a specific disease, but is the first symptom of osteomalacia, and, under certain conditions, the only one in those cases in which this disease is arrested and cured. They consider such cases to have been the basis on which it has been classified as an independent disease. According to Rychner, Nessler, Anacker and Lemke, pica and osteomalacia are two wholly distinct conditions due to specific causes. Nessler maintains that brittleness of the bones, although common in osteomalacia, rarely occurs in true pica. Rychner and Anacker have observed the existence of a complication of the two conditions; while Lemke points out that osteomalacia is only associated with pica when an animal, already suffering from the latter, is allowed to take cold or develop a rheumatic condition. Haubner-Siedamgrotzky, Spinola

and others are of opinion that pica is a primary disease, frequently combined, in its final stages, with osteomalacia.

The older theories as to the relations between pica and food are unreliable. It has been attributed to malnutritious or indigestible food, hay with hard stems, or cut too late, or musty, or which has been exposed to the action of lye ; or an excessive amount of straw ; or grazing on marshy or peaty meadows, on sour plants, such as juncaceæ, equisetaceæ, cyperaceæ, and varieties of rumex ; on too much boiled food or grains ; sour food ; too little or too much rock-salt ; and on feeding exclusively restricted to potatoes or roots. Haubner goes so far as to assign pica entirely to the action of certain individual plants, namely: spicknel or hart's-root, meum athamanticum, achillea millefolium, alchemilla vulgaris, etc. This hypothesis is, however, baseless, and has been refuted by certain of Lemke's experiments. That feeding on equisetum has not caused pica has been fully proved by Kleinpaul.

Observations upon pica were made by Nessler in the Black Forest in 1861, 1867 and 1868, who found, upon an analysis of the hay and water, that the soda-salts were consistently lacking in the food of the affected animals, and, therefore, concluded that their absence played an important part in the origin of the disease. His experiments, however, with fodder deficient in phosphate of lime were undertaken with the object of determining the origin of osteomalacia and not of pica.

Definition.—Pica or licking disease is a peculiar morbid condition which affects cattle more frequently than pigs, horses or goats, and manifests itself chiefly in an irresistible inclination to lick, gnaw and, if possible, eat all kinds of objects even of the most disgusting kind. It is a chronic disease, leading to disturbance of digestion and nutrition, to cachexia and finally to death.

Etiology.—1.—The principal cause of pica is to be sought in the peculiar properties of the soil upon which the food is grown. It may be assumed with certainty that the greater number of cases of pica are intimately connected with certain forms of mal-nutrition, arising from an insufficient supply of nutrient salts. What the exact nature of the change in the food may be, and precisely which of the salts it is whose absence gives rise to the disease, are questions not as yet

determined. Nessler's investigations particularly point to a deficiency of alkalies in the food.

Unquestionably the cause of pica will be found in a want of some vitally necessary food, which may not, in all cases, be of the same character. A lack of nutrient salts in the food leads above all to an abnormal condition of the nature of the secretions. This may be brought about by an affection of the digestive organs—such as intestinal catarrh—or also probably by osteomalacia. There will be an instinctive effort on the part of the animal affected to improve this morbid condition, and, in addition to the ordinary feelings of hunger and thirst, there will be an unusual desire for certain nutrient salts. First there will be a natural effort at self-cure, but this soon passes away, and then pica supervenes in its fully developed form.

2. In some cases pica must be regarded as a disease of the intestinal canal. This applies particularly to those cases in which animals, living under healthy conditions and having the best possible food and care, gradually waste away and die. The assumption that the originating cause in these cases is a chronic dyspepsia or intestinal catarrh, is supported by the fact that a treatment for these maladies has frequently been the means of curing pica. The old theory of "an excess of acids in the primæ viæ being one of several of the causes of pica," shows that it was regarded as a gastro-intestinal affection.

3. Tscheulin and Spinola in particular, have looked upon pica as a nervous disease. Tscheulin considers pica to be a depraved condition of the sense of taste, and therefore discusses it in the chapter on nervous diseases. Spinola and others consider that it is due to an affection of the sensory nerves of the stomach and to a certain derangement of the pneumogastric nerves. Lemke has recently defined the pica of cattle as "A chronic disease of which the origin is to be sought in nutritional disturbance, which is induced by an insufficient supply of phosphorus and commences with a primary affection of the central organs and the nerves which control nutrition." It is certain that the nervous system becomes altered in this disease, and that the nerves of taste are irritated in a peculiar way; but it is extremely doubtful whether any case of pica is due to nervous disturbance. In accordance with the teaching of Haubner-Siedamgrotzky, it might be correct to say that some of the cases of pica are caused by peculiar conditions of nervous irritation.

4. Infection and heredity were formerly regarded as causes

of pica, but on insufficient grounds, as more scientific investigations have proved.

5. Imitation has been frequently given as a cause of pica. But although the persistent licking and gnawing of objects is a vice which, like crib-biting, may be copied from an animal in the same byre, it is impossible to look upon imitation as a factor in the causation of a disease so deep-seated and far-reaching in its effects as pica.

6. Bad stabling is considered by Haubner and others to be a direct cause of pica. The term includes insanitary conditions, irregularity in feeding, and the absence of proper care and attention. But as Lemke justly points out, the disease is as common in the best stables as it is in the worst, and thus the hypothesis should only be accepted with great reserve.

7. With regard to the etiological relation between osteomalacia and pica, the most widely divergent views prevail. Our own opinion on the subject may be summarised as follows :

In many cases osteomalacia is the cause of pica. They are, however, quite distinct, and, although under certain circumstances they may be combined, either disease is to be met with separately. It is by no means improbable that, when we consider the close relationship between these two forms of disturbance of nutrition, we shall find that they may both originate from the same cause.

Occurrence.—Bovine pica occurs sporadically as well as enzootically, and spreads widely in some years. It is a permanent resident in certain districts, especially in the Black Forest and the Erz Mountains. Pica-ridden farms in the Black Forest are known as Hinsch-, Semper-, or Darrfarms (the three words signifying "emaciation" or "wasting-away"), and as early as 1665–1721, Camerarius, in his *Ephem Nat. Cur.* called the affected farms "*villæ tabeficiæ.*" According to Nessler, these farms are always on granite, and never on gneiss, which difference he explains by the fact that gneiss is much more liable than granite to become decomposed by the action of the weather, and consequently it yields up its salts, particularly those of sodium, more readily to the soil and the crops grown upon it. Granite furnishes a coarser-grained and more porous soil. The disease often continues on these farms as long as twenty years, if the animals are not removed to another place. On other farms it appears only during dry seasons, when food is scarce. Haubner

and Spinola state that in some places the presence of the disease makes it nearly impossible to rear calves, and even imported cows are attacked by it after two or three years' residence. Pica is specially prevalent in poor districts, on small farms, and among stall-fed animals, towards the end of the winter, when food becomes scarce. Cows in an advanced state of pregnancy, and those which give abundance of milk are more liable to contract the disease than others, probably because they need a larger supply of nutrient salts. Bullocks are not so often affected as cows. Young animals, especially calves after weaning, suffer from pica, symptoms of which Lemke occasionally saw among sucklings.

Within the last few years pica has become very prevalent among cattle on the Johannisburg moorlands of East Prussia. The disease especially attacks calves and young cattle, and on certain farms is of so severe a nature that it is almost impossible to rear a single calf. It occurs only on those farms where the hay is cut from marshy or peaty ground. As long as the animals graze over wooded or fallow land they remain healthy, but some four to five weeks after they are stalled and fed on hay, the first symptoms of pica appear. On the other hand, cattle suffeiing severely from pica recover quickly when again put out to grass. The reclamation of the moorland has not eradicated the disease ; for neither drainage nor the removal of the topsoil, nor dressing with kainite and Thomas' slag, nor the sowing of the best grasses, has stamped out pica. Kleinpaul says that it has affected animals fed on the very best hay, and this is confirmed by experiments with fodder made at the Berlin Hygienic Institute (Ostertag and Zuntz). Full-grown cattle, when taken to the Johannisburg district, develop the disease in the second year.

From these facts we infer that the fodder obtained from certain marshlands lacks something which is essential to the proper food for cattle. Or it may be that grass grown upon such soil, when made into hay, undergoes a certain change in its nutritional properties which brings on pica.

Symptoms.—At the beginning of the attack, and for some time longer, there is no fever. Lemke divides the course of the disease into a first, or apyretic period, and into a second, or febrile period. The first perceptible disturbances consist in a somewhat diminished, or slower consumption of food, and in indolent and infrequent rumination. The appetite soon

becomes capricious, and symptoms of pica make their appearance. Unless compelled by hunger, the animal will eat by preference large quantities of straw from the floor, and when pasturing will, contrary to its normal taste, prefer highly manured rank grass, and young sprouts of trees and shrubs, or in the stall try to approach the attendants in order to lick* their clothes with peculiar eagerness. Day and night the animal continues to lick more and more persistently both bars, walls, woodwork, etc., frequently with loud clicking movements of the tongue, and will generally gnaw these objects as well, and even its iron neck-chain. At the same time there is increasing restlessness and excitement, and often abnormally great sensibility over the course of the vertebral column, especially in the lumbar and sacral regions. According to Lemke, these signs continue as long as four months, but vary somewhat in their intensity. At this period, as a rule, the state of nutrition is not much disturbed, nor the decrease of milk considerable, and the licking and gnawing can almost always be stopped by threatening the animal. The symptoms soon become aggravated, particularly the desire to gratify their depraved appetite, in consuming soiled litter, dung, decayed wood, old rags, ropes, leather, and especially earth, loam, sand, substances containing lime and clay, fragments of mortar and bricks, and any tainted water in preference to pure water. It even happens that affected cattle eat each other's hair. (Utz).

In addition to decrease of appetite, in consequence of the constant licking and gnawing, which prevent the animal from obtaining the necessary time to feed, refusal of good food, and consumption of all kinds of indigestible and disgusting things, rumination becomes shorter and less frequent, and the movements of the rumen grow weaker and are sometimes temporarily suppressed. The milk becomes poor in fat (Lemke) although its quantity may continue normal for a long time (Leytze). The dung generally appears dry and even hard,

* Even perfectly healthy cattle, especially if stall-fed, will often show extraordinary obtrusiveness and desire to lick the clothes of strangers going into the stall. These animals may also take up and swallow very curious things, without being in any way affected by pica. Markeinche relates a strange case in point of a healthy cow which had never been ill, and which was one of the best feeders, devouring one after another, a pair of cotton stockings, cheese wrapped up in a cotton cloth, a waistcoat with metal buttons, a child's merino frock, and a woman's calico jacket, with the exception of the sleeves. Similar examples are found in Gurlt's *Pathological Anatomy.*

and covered with considerable masses of ropy mucus, or it may be semi-fluid and fœtid. The urine is usually acid and sometimes contains albumen. At this period the temperature shows a rise of from 1° to 3° F.; and the rate of the pulse is from 70 to 80 a minute. The pulse is small and the arteries are contracted, but respiration does not seem to be much disturbed.

During the further course of the disease, the animal becomes quickly emaciated, almost to a skeleton, and especially after calving. The visible mucous membranes are pale, the hair lustreless, rough and erect; the skin, which is abundantly covered with dandruff, is dry and hard; and if we pinch up a fold of the tense skin over the ribs, it will emit a crackling sound, and when we let it go, the fold will become effaced very slowly, showing that in this "hide-bound" condition the skin and subcutaneous tissues have lost a good deal of their elasticity. With increasing weakness and marasmus, the animal generally stands with its feet placed under its body and its back arched; it occasionally grinds its teeth, and its movements, especially those of the hind-quarters, are stiff, narrowly limited in extent and accompanied by crackling sounds of the joints. Finally, the animal is unable to keep on its legs, refuses nearly all food, the sounds of the rumen cease, and it dies from exhaustion.

Course.—The disease when left to itself is chronic, and its duration, according to Lemke, may extend from six months to two years. Spontaneous recovery may take place in from three weeks to four months (Lemke), especially in the early stages of the disease, by appropriate change of food or pasture, or changing from stall life to grazing in the spring, and *vice versa* in autumn. If preventive measures are not taken the case will generally end in death.

Anatomical Conditions.—In cases of slaughter, which are naturally those of an early stage of the disease, there is usually little to be seen, beyond slight emaciation and catarrhal changes in the mucous membrane of the alimentary canal. In animals which have died of the disease, we find extreme general emaciation; great deficiency of fat; a gelatinous condition of all the adipose tissue, which is frequently of a dun colour; the muscles greatly atrophied, flabby and pale; and the blood deficient in quantity, pale in colour,

thin and clotting but feebly. We sometimes find slight transudations in the thoracic, abdominal and pericardial cavities, and a more or less severe catarrh of the mucous membrane of the gastro-intestinal canal. The cutis and subcutis, being in sympathy with the chronic disturbance of nutrition, are dry and hard. The changes which are said usually to occur in the bones, belong most probably to osteomalacia, and not to pica.

Prognosis.—There is no prospect of recovery, even in changing the food, if general emaciation and digestive disturbances are present. In slight cases a favourable prognosis depends on the possibility of removing the cause, especially by changing the food or pasture land. If the statements of Lemke with respect to the excellent results of apomorphine in the treatment of pica be confirmed, prognosis will be greatly improved, even when a change of food is impracticable.

Treatment.—It is first essential to discover the cause. Prophylaxis, as a rule, will be best carried out by improving the meadows and pastures by artificial manures, particularly nitrate of soda. But the experiments in the Johannisburg fens have shown that such improvement of the pastures is by no means always attended by beneficial results. We know from experience that the best and most natural means for combating the disease is to make a thorough change in the food, which we may effect either by obtaining food from districts that are free from pica, or by removing the affected animals to these places. As insuperable difficulties would usually attend the realisation of these measures the efforts of the veterinary surgeon are generally restricted to correcting as far as possible the faulty conditions of fodder. This is best done by the addition of nitrogenous and leguminous foods and common salt. In certain isolated cases a change in the drinking-water has been attended by beneficial results. As an instance, pica completely disappeared from an estate in Posen after the animals were watered from a neighbouring estate which was free from the disease.

From ancient times attempts have been made to improve digestion by the administration of weak hydrochloric acid, small doses of common salt, bitters and aromatics, and in instances of excessive acidity, by antacids and absorbents (lime water, chalk, magnesium carbonate, sodium bicarbonate, etc.), which treatment is particularly applicable to cases which have

their origin in digestive affections. When osteomalacia is the cause, phosphate of lime is indicated. The administration of nauseous matters, such as garlic and stinking animal oil, has long been tried. Feser recommended the experimental use of hydrochlorate of apomorphine, which has been successfully used by Hackl and Reindl. In more recent times, Lemke has proclaimed it as the sovereign remedy for pica, and supports this statement by the results of a large number of experiments. Out of 226 cows, 21 oxen and 141 calves which Lemke treated by this means, all the animals without any exception recovered in a few days, and took only from eight to fourteen days to regain their appetite. In fresh cases he gave three grains, and the same dose to calves. If the exciting cause be removed, this treatment will bring about a permanent cure ; otherwise the effects of the apomorphine will not last longer than three to five months. Lemke further states that in localities where pica is enzootic and the cattle have, in consequence, to be changed every year or two, they may be kept for three years in a comparatively good condition if treated with apomorphine four times a year. For cattle which have been suffering for some time, he used apomorphine subcutaneously in doses of 1½ grains daily, on three consecutive days, and double that amount for those which had recently contracted the disease. The observations of Väth Gassner, Ostertag and Wagenheuser are very favourable to the efficacy of apomorphine in this disease ; but Schuemacher, Röder, Flum and Hafner report failures or disagreeable effects from the use of the drug. According to Kleinpaul (Johannisburg), apomorphine reduces the symptoms for a time, but does not remove the primary disease. His method is to give three doses of 1½-2½ grains a day for six successive days. Calves, however, as he noted, exhibited much excitement, rushing wildly about the byre. He considers that the only sure way of combating pica in Johannisburg is to turn the animals out to graze as early in the year as possible.

Pica in Horses.—True pica is much rarer in horses than in cattle. Usually it is sporadic, and presents only slight symptoms. The cause is generally gastric disturbance, in which case the disease may be successfully treated by stomachics (neutral salts, Carlsbad salts, hydrochloric acid, etc.), or by antacids (lime water, carbonate and bicarbonate of soda, carbonate of magnesium, etc.). Steinmeyer obtained good results in a horse by the subcutaneous use of ¾ grain of apomorphine

in $2\frac{1}{2}$ ozs. of water. The symptoms of the disease, which is found most frequently in foals, consist in persistent licking and gnawing of adjacent objects, or of the animal's own skin, diminution of appetite for normal food, and desire for eating litter and dung. The disease is found most frequently in foals, which may become reduced to mere skeletons. For further remarks upon the eating of sand and earth by cavalry horses during manœuvres, see chapter on Colic (Sand-colic).

The eating of sand, which has been observed in many remount depôts, and which is the cause of "sand colic," may be attributed to play or vice in young horses left to themselves. It is found among those remounts that are left without supervision the entire day in paddocks laid down with sand, which they gradually learn to eat from mere *ennui* and by imitation. The vice can therefore be checked in the majority of cases by stabling the animals and keeping them under strict supervision. The eating of sand is, however, sometimes a symptom of true pica, in which case it may be referred to deficiency of certain mineral substances in the food, and be treated by improving the soil, and by giving common salt, phosphate of lime, etc. We may also try to prevent the consumption of sand by sprinkling it with substances which have a disagreeable taste, such as tar, carbolic water, creolin water, wood vinegar, paraffin oil, etc., and by subcutaneous injections of apomorphine (three-fifths of a grain to three grains). An absolutely reliable remedy for sand-eating does not, however, exist. Foals have another bad habit of eating the hair of the tails of their mothers. This vice has nothing to do with pica. G. Jaeger tells of a cavalry horse which had a passion for eating sparrows, rats and mice, both dead and alive.

Pica in Pigs.—According to Spinola, pica affects pigs in the same districts in which it attacks cattle, and is similar in both species as regards its course, duration and termination. It is stated that the animals eat by preference rotten wood and bark. Jansen states that symptoms of pica are seen in osteomalacia. Treatment consists in change of food, and in the administration of soluble bone meal. Emetics (tartar emetic, hellebore, ipecacuanha, veratrine, etc.) may be used experimentally.

The devouring of newly-born pigs by sows has as little connection with pica, as the eating of the after-birth by cattle. It does not seem to be the outcome of play or vice, but appears in

the majority of cases to be due to temporary mental aberration, and occasionally to digestive disturbance, as constipation, for instance. Block recommends an emetic as a simple and reliable remedy. After removing the young ones, milk containing tartar emetic and powdered hellebore root (of each eight grains) may be administered to the sow, which by this means will become tranquil, and will receive her litter kindly. An aperient (calomel) and green food may also be prescribed. As a precautionary measure young pigs in France are frequently sprinkled with some bitter substance, as gin.

WOOL-EATING OF SHEEP (*Ovine Mallophagia*).

Etiology.—The eating and gnawing of wool by sheep is an affection closely allied to pica of cattle, and is of great economic interest, owing to the destruction and loss of wool entailed by it. It has been cited as a cause of death in lambs. Opinions on the origin and nature of wool-eating are as various as those regarding pica. There are, however, two theories which have received more support than the rest. Some authorities are of the opinion that the disease is purely the result of imitation, others that, like pica, it proceeds from a condition of mal-nutrition. It would be difficult, in the present state of our knowledge, to decide exclusively in favour of either hypothesis. It is probable that both causes are in operation at once.

1. The *imitation* theory has its chief partisan in Spinola, who assumes that *ennui* prompts the first animal to eat wool. The following facts seem to indicate that the vice arises from imitation:—Firstly, only one sheep as a rule begins to eat wool. Secondly, Spinola was able to see the vice only in certain pens on the same sheep farm. Thirdly, the spreading of the disease in a herd was checked or entirely stopped by a strict separation of the wool-eaters (Spinola and Burmeister). Fourthly, the wool-eating occasionally begins even before the sheep are housed for the winter.

2. For many years the affection was ascribed to ***deficient and faulty feeding***. This is a theory particularly advocated by Lemke, who believes that his experiments prove that it cannot be caused by imitation. It is especially frequent during winter, when fodder is insufficient in quantity, and largely supplemented by potatoes, potato "mash," and other food poor in

nitrogen; but it disappears with a change of food. In lambs it is ascribed to deficiency of milk, starvation of the ewe, or to an abnormal condition of the ewe's milk. May states that on a sheep farm the lambs became wool eaters on account of not getting enough milk from the ewes; but that they gave up the vice as soon as they experimentally received an addition of cow's milk. Haubner-Siedamgrotzky attributes it also to peaty, sour, wet pasture, and a deficiency of common salt.

Occurrence.—Wool-eating is found principally in finer, and especially in the merino breeds, and, according to the unanimous statements of all authors, only as long as the sheep are stall-fed during winter, namely, from autumn to spring, but rarely while they are at pasture. The affection frequently appears within from eight to fourteen days after the sheep have been housed, but disappears just as quickly when they are turned out to grass. Even putting them on corn during the winter has an immediate good effect. According to many agriculturalists wool-eating is by no means uncommon, though more prevalent in some years than in others, even where the food be the same.

Symptoms.—In cases of wool-eating by lambs, the animals are said to begin at the age of from two to six weeks, by gnawing the wool of their mothers, preferably that of the legs, belly and tail. At first only a little wool is gnawed off in play; but the desire gradually increases, and the lambs finally gnaw other sheep. On the whole, they appear healthy, without any marked disturbance of appetite or growth. The ill-results are evident only when the lambs, as sometimes happens, consume a great deal of wool, especially if it be long and coarse. They then become emaciated, are backward in development, constipated at times, and may even die with symptoms of gastro-intestinal inflammation. On *post mortem* examination, besides signs of inflammation, especially of the abomasum, we find pellets of wool varying in size from a hazel-nut to a walnut, and quantities of other wool, which are supposed sometimes to get into the pylorus and obstruct it.

If wool-eating appears in a flock of yearlings and older sheep, only one animal as a rule begins to eat the wool of the others, apparently as if seeking for forage in the fleece. It is stated that wool soiled by urine and dung is preferred. The first wool-eater often gnaws several sheep, but finally confines its attention to one. Others, one by one, soon join

in and help him in working on the selected sheep. As a rule, they do not begin on another until they have cropped the firs one bare. With the increase in the quantity of wool-eaters, the number of those selected to be clipped becomes greater and greater, with a corresponding loss of wool. The wool-eaters remain apparently healthy, free from fever and loss of appetite, and their rumination is not disturbed. Occasionally some of them suffer from constipation, dryness of the wool, etc.; although wool-eating does not lead to well-marked cachexia and marasmus, ending in death, as in pica of cattle, and it does not set up osteomalacia or osteoporosis.

If the vice be but slightly developed, it is practised only by day, and generally in the intervals of feeding; but if it be in a severe form, it may be carried on also by night and in dark stalls (Lemke). Cases in which sheep nibble their own wool are extremely rare; consequently, this affection is not liable to be confounded with scab and certain eczemas, which are accompanied with pruritis.

Prognosis.—Although mallophagia is a much milder disorder than bovine pica, death may occur from it among lambs. The remarks made on pica with respect to change of stalls, food and pasture, and the use of apomorphine, also apply to the prognosis of wool-eating.

Treatment.—Change of food and a liberal supply of nutriment are the first consideration, especially with lambs, and grazing is preferable to stall-feeding, if the weather permits. May recommends that the lambs be separated from the ewes after the third week from birth, but should be allowed to suck several times during the day.

No time should be lost in separating the first wool-eaters and the sheep that have been cropped, from the remainder of the flock. The administration of common salt, alkalies, stomachics, etc., may also be tried. Spinola recommends that the stalls be kept dark during the intervals of feeding in the day, a measure not likely to be of much use in advanced cases.

Lemke recently obtained equally good results in cases of ovine mallophagia, as he did in those of bovine pica, by the subcutaneous use of hydrochlorate of apomorphine; he cured eight hundred wool-eaters by this remedy, without a single failure. He uses the same doses (1½ to 3 grains) for sheep as for cattle. The curative effect of this drug is said to last for

four months. These experiments of Lemke, if borne out by subsequent investigation, put us in possession of a valuable specific in a department where, up to now, drugs have been of little use to us. In any case, the treatment should be tried.

The Plucking out of their own feathers by Birds.—This vice is produced by *ennui* and desire to play. It is practised chiefly by parrots and canaries, and sometimes by cocks and hens and other poultry, and is generally incurable. We have unsuccessfully tried external applications of the tinctures of veratrin, asafœtida, aloes and gentian and other bad tasting fluids, and believe we have obtained cures in certain cases by the internal administration of apomorphine (¾ grain in 3½ oz. of water). This treatment should be supplemented by a change of diet and the birds kept as much as possible in the open air. Poultry should be given green food, animal food and blood; parrots boiled maize, hemp-seed and wheaten bread. This plucking of feathers must not be confounded with moulting during the course of certain skin affections.

CHAPTER III.

ACUTE CATARRH OF THE STOMACH AND INTESTINES IN HORSES.

(*Gastro-enteritis catarrhalis acuta—acute dyspepsia.*)

Preliminary Remarks.—In this chapter we intend to deal collectively with acute catarrh of the stomach, acute intestinal catarrh, and the various abnormal conditions of the stomach which are known as indigestion, impaction of the stomach, dyspepsia, weak digestion, difficult digestion, gastricism, gastrosis, status gastricus, gastric fever, etc. In this respect we follow the old routine, which is based on the fact that it is extremely difficult, in the horse, accurately to locate the site and determine the intensity of a disease of the stomach or intestines. There is no doubt that in our domestic animals, an acute catarrh may be confined either to the stomach or intestines, or may be restricted to certain parts of the intestines. Also, there are many stages between the physiological hyperæmia of the mucous membrane of the stomach during digestion, and intense inflammation of that membrane. As vomiting, which is a characteristic symptom of gastric catarrh in other animals, is almost always absent in horses, and as the diarrhœa of intestinal catarrh is often not apparent in them, owing to the great length of their intestines, it is hardly possible in horses to distinguish between gastric catarrh and intestinal catarrh, which diseases very often succeed each other, or are simultaneously produced by one and the same cause.

We are also in want of reliable diagnostic means to distinguish between individual degrees of intensity. Formerly the mildest form of gastric disease was called dyspepsia, which was held to mean only a disturbance of the physiological function of the stomach, without any demonstrable changes in the structure of the mucous membrane. In the domestic animals the presence of such a form of dyspepsia may be suspected, but

cannot be proved, although in all probability it occurs in old and anæmic animals, when convalescent after prolonged illness, etc. Unfortunately it is quite impossible to say where dyspepsia ends and catarrh begins, especially as the former, if it be present for a long time, may change into the latter. We have, therefore, refrained from classifying dyspepsia as an independent disease. It is comparatively easy, however, to distinguish between the clinical aspects of a slight gastric catarrh and severe gastro-enteritis. For this reason we have thought it advisable to discuss the two conditions separately, and to deal with gastro-enteritis in another chapter. In actual practice, the veterinary surgeon finds that there are many transition stages between the two conditions, and that he is frequently in doubt as to whether he shall base his diagnosis upon " inflammation " or " catarrh." This is the reason why so many text-books include under one heading both catarrh and inflammatory affections of the gastro-intestinal canal. But it must be remembered that inflammation of the gastro-intestinal mucous membrane constitutes a condition of severe illness accompanied by pronounced symptoms, such as high fever and grave general disturbance, and can be distinguished, not only anatomically, but also clinically from simple catarrh.

Etiology.—The causes of acute gastro-intestinal catarrh, which is a very common disease in domesticated animals, and especially among horses, are partly certain noxious matters that exercise a direct influence on the mucous membrane, and partly factors of a more indirect nature. Special susceptibility to the disease is found in some individual animals, such as weakly, anæmic, badly-fed, pampered foals, very old horses, convalescents from severe diseases, animals whose constitutions have become weakened by relaxing food, and those whose powers of resistance have been diminished by previous attacks of gastro-intestinal catarrh. Some individuals of the finer breeds and those which are accustomed only to the best food, are very liable to contract the disease from slight causes.

Among the direct irritants of the mucous membrane we may mention water, and fodder which is too hot or too cold ; grass which is frozen or covered with hoar frost ; boiled food ; too hot a mash when the horse is carelessly exposed to draughts ; too hot clysters ; unclean or damaged fodder ; bad or dirty water ; the aftermath cut from flooded meadows ; mouldy, rusty or dirty hay ; damp or dirty straw ; mouldy or spoilt oats ; mouldy

bread; fermenting or decomposing food-stuffs, such as rotten potatoes, roots, or fruit; fermenting green-stuff; sprouting grain; mouldy cake; all irritant substances, and especially fodder containing poisonous plants (colchicum, tobacco, digitalis); the improper use of strong drugs and purgatives; sour milk in the case of sucklings; indigestible substances, such as the woody fibres or elastic tissues of plants; an exclusive feeding on maize without salt; and too much corn, beans, or peas. The last-named are not only indigestible in themselves, but they also swell in the stomach. A fruitful cause of gastric disturbance is imperfect mastication of food, especially in young horses when cutting their teeth with difficulty. The gums sometimes become very tender, and mastication painful and imperfect. Young foals are frequently thrown back in this way. Another cause is the giving of too large a quantity of food and water. Over-feeding is generally the result of a sudden change from a light to a rich diet, or the reverse, and especially from one that is easy to one that is difficult of digestion; the too rapid transition from stall feeding to grazing, particularly as this involves an unusual amount of sappy and easily fermenting green-food—especially of clover. It occurs, also, if the horses are allowed to become too hungry, as when the intervals between the feeds are irregular and prolonged or the quantity of food reduced. Irregularity is the most fruitful cause of over-feeding, but it may arise from the animal being ravenous or be the result of its breaking loose in the stable and getting at the fodder. Cases of acute gastro-intestinal catarrh are sometimes seen after feeding on new hay. Strangely enough, among the indirect causes of gastro-intestinal catarrh, over-exertion, and the working of animals too soon after feeding are the most frequent. In these cases the condition is due to unequal distribution of blood, which is drawn away from the organs of digestion to the muscles of locomotion. External cold is another fruitful cause. A sudden lowering of the temperature, or prolonged exposure to a low temperature, induces hyperæmia of the mucous membrane and so creates a condition predisposing to catarrh. Excitement, temper, or training will provoke this, as also very hot or sultry weather or ill-ventilated, damp or dirty stables, as these bring on chills or debilitation.

It sometimes happens that a form of acute gastric catarrh makes its appearance in certain districts as an enzootic disease. This so-called infective gastro-intestinal catarrh is caused by the entrance into the alimentary canal, of micro-organisms about

which we possess but little information. In its initial stage, it is difficult to distinguish this condition from the others.

Besides these cases of independent acute gastro-intestinal catarrh, we find a symptomatic form of this affection in the course of different febrile diseases, such as pleuro-pneumonia, distemper, puerperal fever, petechial fever, etc.

Post-Mortem Conditions.—As the primary acute catarrh is rarely the cause of death, a *post-mortem* examination is only possible in a case of secondary catarrh. Even then we may have a negative result, because the inflammatory changes that are present during life sometimes almost disappear after death.

1. *Appearances in the Stomach.* The true mucous membrane of the stomach (that of the pyloric half) shows signs of catarrh, inflammatory swelling with considerable redness and severe congestion, which is sometimes diffuse and at other times in patches.

These hæmorrhages, which vary in size, are sometimes punctiform, lenticular, or even larger, and give the mucous membrane a dotted, sprinkled oı spotted appearance. The mucus is increased in quantity, is sometimes glairy, at other times purulent; may be stained with blood in consequence of slight hæmorrhages, and may contain cellular constituents, mucous corpuscles, leucocytes, and desquamated and decomposing epithelial cells. Microscopical examination of the affected mucous membrane reveals mucous degeneration of the columnar epithelium of the excretory ducts of the glands, considerable granulation of the cardiac glands, cellular infiltration of the inter-glandular connective tissue and submucosa, with swelling and desquamation of the endothelium of the lymph vessels.

2. *Appearances in the Intestines.* The pathological changes in the intestines are similar to those in the stomach. The mucous membrane is inflamed, swollen and loosened, in consequence of the serous infiltration of the submucosa; the muscular wall of the intestine contains an abnormally large amount of fluid, is consequently tender, and easily lacerated: The inflammatory hyperæmia is sometimes narrowly limited in extent; at other times it involves large portions of the intestine, but seldom invades the entire intestinal canal. Sometimes the hyperæmia and consequent œdema is confined to the intestinal villi, which become enlarged and elongated, and project in a striking manner into the lumen of the intestine,

or the congestion takes the form of an area around the more or less swollen Peyer's patches. In consequence of the swelling, these follicles sometimes project above the mucous membrane in the form of light grey nodules, which vary in size from a grain of millet to a hemp seed, and are surrounded by a red area, in which case the name of " follicular catarrh " has been given by some authorities to the condition. If these inflamed follicles become destroyed by suppuration or by attrition, small crateriform ulcers will be developed on their respective sites. These losses of substance, which may be called follicular ulcers, must not be confounded with true intestinal ulcers. In severe cases we may find diffuse epithelial desquamations (catarrhal erosions). The affected mucous membrane excretes a fluid which is rich in epithelium, and which may be serous, mucous, or purulent, according to the quantity of its cellular constituents. When hæmorrhages are present, its usual light grey or buff colour may become tinged with red. Sometimes the slackness and desquamation of epithelium is particularly abundant (desquamative catarrh).

Symptoms of Acute Gastric Catarrh.—The first and often the only symptom is irregularity of the appetite, which may be quite lost, or the animal will either eat slowly and less. The appetite is frequently very irregular, at one moment quite normal and at another disappearing altogether. The animals are frequently dainty in their food; they constantly refuse certain kinds of fodder and show a preference for others, such as rough fodder and straw. Symptoms of perverted taste, as in pica, such as the eating of dirty straw, may make their appearance. Thirst decreases and many animals refuse water altogether. On account of irritation to the terminal branches of the pneumogastric nerve, the animal may yawn uninterruptedly for hours, or have ceaseless running at the nose. In the horse, overloading of the stomach may give rise to eructations and actual vomiting, without any great danger to the patient, and without signs of severe colic. We learn from veterinary literature that such cases of uninjurious vomiturition are by no means as rare as is generally supposed, particularly in mountainous districts where horses at grass sometimes consume a large quantity of the leaves of white hellebore. But it must be borne in mind that the upper air passages may become choked with the vomited food and thus give rise to asphyxia (Zschokke).

As a rule, the mucous membrane of the mouth presents important changes, and is either highly congested or pale. It is sometimes of a dirty yellow or livid colour. At first it is often very dry, and later on may be covered with a greasy, pappy, lather of frothy mucus, which has an insipid sweetish smell, or we may find an accumulation of glairy mucus, alkaline in reaction. Also, a swelling of the submucous venous network on the hard palate is often observed. The tongue is frequently coated, on account of the diminished consumption of food. Acute gastric catarrh is very often complicated with catarrh of the entire buccal membrane. At first the abdomen is not much affected, but later on it becomes empty and tucked up. Peristalsis may be normal, or may be decreased from the very beginning of the attack. The fæces are often balled, dry, covered with a shining crust, are passed in small quantities at a time, often at short intervals, and frequently contain undigested particles of food. Colic is absent, as a rule, and the urine is nearly, if not quite, normal.

The mental condition varies according to the severity of the affection and individual idiosyncrasy. The animal is dull, depressed, easily fatigued, sweats with the least exertion, and breathes with difficulty if put to work. In many cases there is no rise of temperature; it is seldom very high. Usually the temperature of the rectum is not increased more than one degree Fahrenheit, and the pulse not more than eight to ten beats. If fever be present, it generally lasts for only a few days. In very rare cases, rigors may appear as a preliminary symptom. On the other hand, we often find unequal distribution of temperature, as may be shown by coldness of the ears and limbs, and by congestion of the conjunctiva.

Symptoms of Acute Intestinal Catarrh.—Contrary to what is the case in acute gastric catarrh, the appetite may be normal in acute intestinal catarrh. It is, however, as a rule, more or less disturbed, especially in the later stages of the disease. There is frequently great thirst if diarrhœa be present. The general health may remain unchanged, without any appearance of fever, although the total constitutional disturbance is usually more severe than in catarrh of the stomach. The chief symptoms arise from the intestine. Peristalsis becomes more active; the intestines give out rumbling and gurgling sounds which are often audible at a distance; but at certain times the intestinal

movements may be stopped, and no sounds given off. The abdomen is occasionally tympanitic, and, in that case, painful on palpation. The dung is at first in the form of large soft balls, frequently covered with glairy mucus, or enveloped, as in a net, in dirty white or intensely yellow membranous, croupy masses (*Proctitis*). It usually contains a lot of undigested food, and emits an acid or putrid smell. Occasionally, after passing fæces, the animal discharges a greenish-yellow fluid, which soils the parts adjoining the anus, tail and inner surface of the thighs. Flatus, which generally has a fœtid smell, is often passed during defecation, and also independently of it.

Later on we see diarrhœa, which is a very important symptom for the diagnosis of intestinal catarrh. The fæces, which at first were soft and moister than usual, assume the consistency of cow-dung, and finally become thin and quite fluid. They emit a remarkably disagreeable acid smell, and are sometimes passed involuntarily through the gaping anus, and under conditions of great weakness (colliquative diarrhœa). The reaction of the fæces is usually acid (from the presence of fatty acids), which is a fact of no importance in diagnosis, because they often have this reaction under normal conditions. In some cases there may be no diarrhœa. If the catarrh be confined to the small intestine, the excess of fluid in the chyme passed onwards by it may become entirely absorbed in the large bowels, and consequently the dung may be discharged in balls. We should bear in mind that the presence of diarrhœa does not always imply the existence of a catarrhal intestinal affection. Apart from the fact that the fæces may be very soft when a horse is fed on green food, temporary diarrhœa may appear in consequence of increased peristalsis from reflex action, or as a result of temporary hyperæmia, due, for instance, to chill.

The urine, which suffers little or no change during gastric catarrh, manifests various abnormal conditions in acute intestinal catarrh. If diarrhœa be present, it becomes diminished in quantity, with a corresponding rise in specific gravity, often changes in colour, loses its sediment and becomes clearer. The amount of indican contained in it is increased. The most noteworthy point about the urine is, that it is abnormally rich in phosphates, only traces of which are found in heathy urine. Its reaction, which in normal conditions is alkaline, is neutral or acid.

Acute gastric catarrh in horses is sometimes accompanied by a pimply rash, while dullness of sight (amblyopia) may also

occur. This is probably due to the absorption into the system of fermented and decayed matter.

Duration and Progress.—The majority of cases of acute gastro-intestinal catarrh which are accompanied only by slight or moderate fever, recover quickly, even in a few days, and seldom continue beyond a week. The fever gradually subsides, the appetite improves, the evacuations diminish in frequency, and the fæces become more solid. Occasionally the diarrhœa is followed by slight constipation. The return of the urine to its normal alkaline condition indicates improvement. In severe cases the catarrh becomes chronic or increases in intensity; the diarrhœa permanent, dysenteric and fluid; more or less severe and frequent colic supervenes; emaciation sets in, the flanks become tucked up; the coat rough and staring; and the temperature of the limbs, cold. These symptoms are combined with rapidly increasing weakness and depression. The animal stands dull and listless in its stall, and shows its debility and exhaustion by lying down, and by frequently lifting up and putting down its hind legs alternately, so as to avoid placing weight on them. Appetite is then entirely lost. If recovery takes place in such a case, convalescence will be greatly protracted. Death will be the usual result under such conditions, especially with debilitated and old horses.

Differential Diagnosis.—It is hardly possible to mistake gastro-intestinal catarrh for other affections of the intestines, if no fever be present. As it is often difficult to decide whether the gastro-intestinal catarrh is independent or whether it is a symptom of a graver disease, we can only diagnose the former with certainty when we are sure that the latter is absent. Difficulties may also arise in distinguishing a severe case of gastro-intestinal catarrh from inflammation of the stomach. Here the diagnosis will depend on the further course of the disease, the degree of the fever, the gravity of the general symptoms, and the consideration of their causes. If, during the course of an acute catarrh of the large intestine, follicular ulceration becomes developed, it will in some cases be impossible to distinguish the supervening dysenteric diarrhœa (which has also been called catarrhal dysentery) from true infective dysentery. Here also, the further course of the disease, the history of the case, etc., will be the data for diagnosis. There are also some important points with respect to the localisation of the catarrh on different parts of the intestinal canal:—

1. Where the disease is confined to the small intestine, jaundice is seldom absent. Owing to the occlusion of the orifices of the gall-ducts by the swelling of the mucous membrane or by catarrh reaching the ducts themselves, there is no channel of escape for the biliary secretion which becomes reabsorbed into the system. If jaundice be absent, we may infer the presence of catarrh in the small intestine by the affection of the stomach, and by the remarkably active peristaltic movements of the small intestine, which will be manifested by loud rumbling sounds that can be best heard in the region of the false ribs and flanks.

2. If the large intestine be the chief seat of the disease, severe colic and diarrhœa will appear. A very characteristic symptom, particularly of the posterior portion of the large intestine (rectum and its adjoining parts), is the fact of the fæces being covered by membranous or retiform slime, consisting of mucus and epithelium, which covers the outside of the dejecta only. In well-marked catarrh of the rectum, the fæces are passed with abnormal frequency and only in small quantities at a time, even in single balls, defecation being accompanied by violent straining, groaning and arching of the back.

Treatment.—The prophylaxis of acute gastro-intestinal catarrh consists in attention to hygiene. Treatment in the first place should be of a dietetic nature, and suffices in many cases. We should give small rations of easily digestible food and, especially in cases brought on by overfeeding, should allow the horse to fast for some time before giving food. We should give only "luke-warm" water; should keep the animal comfortably clothed and have him wisped or hand-rubbed all over several times a day. Another important point is to take the patient for a short walk in the open once or twice every day. The medicinal treatment depends on the indications.

1. In impaction of the stomach the first thing to do is to remove the excess of food as quickly as possible, which we may by a dose of tartar emetic 1½ dr. and sulphate of soda 10 oz., or by giving several small doses of calomel (up to 45 grs.). Care has to be used in the administration of physostigmine (1½ gr.) and arecoline (1¼ gr.), as they may cause rupture of the stomach, when that organ is greatly overloaded and distended.

2. In cases of abnormal fermentation in the stomach and intestine, we strongly recommend dilute hydrochloric acid (2·4 drams in the drinking water as a dose). Common salt (4 dr.

to 1 oz., and bicarbonate of soda (½ to 1 oz.) also render good service. In cases of gastro-intestinal catarrh with flatulence and acidity of the intestinal canal, we have had good results with hyposulphite of soda. Creolin, which is a good antizymotic, can be given in doses of 3 to 6 drams, (creolin 2 oz.; glycyr. rad. 4 oz.; Extr. glycyr. et ceræ flavæ q.s. to be made into 6 balls, 1 or 2 to be given daily). Röder recommends xeroform as an intestinal disinfectant (2½ to 4 drs.).

3. If dyspepsia is present, we may try (besides hydrochloric acid) the true stomachics, such as pepsin, and, most important of all, the alkalies (1 to 2 dr.), common salt, Glauber's salts. sal-ammoniac (sulphate of soda, 10 oz.; chloride of sodium, 4 oz.; chloride of ammonium, 2 oz.; a tablespoonful at each meal), Then there are the vegetable stomachics, mustard (½–1 oz. powdered and given as an electuary), pepper, ginger, tincture of veratrum (½–1 oz.), acorus calamus (¾ oz.), rhubarb (pulv. rad. rhei. 225 gr.; sodii bicarb. 150 gr.; pulv. rad. glycyr. 150 gr.; pulv. rad. althrææ et aqu. font. q. s. f. electuar. to be given in 3 doses), aloes (1½ dr. in a ball). All the bitters may be employed (absinth, gentian, etc.), as well as the volatile oils (anise, cumin, fennel, juniper berries).

4. In cases of diarrhœa, when only small quantities of fœtid fæces are passed, we recommend at first, for the clearing out of the intestinal canal, small doses of calomel (30 to 75 gr.), which can be given even when follicular ulcers are present. We should then try to stop the diarrhœa by dietetic means, such as dry food, parched flour and parched oats, with mucilaginous adjuncts. If we do not attain our end by these means, we may resort to astringents, such as oak bark, cinchona bark (1¾ oz.), sulphate of iron (2 dr.), or alum (¾ oz.), tannin (3–4 dr.), tannoform (¾ oz.), tannalbin, tannopin (very costly). In severe cases sugar of lead (75 grs.), or even nitrate of silver (7 to 15 grs.) in a pill, with Armenian bol dissolved in a large quantity of distilled water. Opium is undoubtedly the best remedy in severe diarrhœa, and may be given in a ball (75 to 300 gr.), or as tincture of opium (1 to 3 oz.) as a single dose in a mucilaginous decoction. In extreme cases it is as well to give opium at once, as its effect is prompter and surer than that of the other astringents—tannin, for instance. We have given as much as 10 oz. ourselves without any unfavourable consequence. Trasbôt advises an emulsion of 150 gr. of camphor, 150 gr. of asafœtida, the yolks of 2 or 3 eggs, and a quart of rice water, to be given twice daily. Röll strongly recommends liquor strychninæ,

(1 to 2 drs.); Schindelka prefers tannopin (2 to 4 drs) as an astringent; while Foth recommends tannoform (3½ to 5½ drs. pro diem).

5. With cases of catarrh of the large intestine involving the rectum, we should supply local treatment by means of enemas. Even with ordinary diarrhœa, enemas of nitrate of silver are often much better than giving this drug by the mouth. Enemas are particularly beneficial in chronic cases of intestinal catarrh. In catarrhal proctitis we may advantageously employ 1 to 2 per cent. solutions of alum or tannin.

CHRONIC CATARRH OF THE STOMACH AND INTESTINES IN HORSES.

(*Gastro-enteritis catarrhalis chronica, or chronic dyspepsia.*)

General Remarks.—We have already explained that it is impossible to make an exact differentiation between chronic gastric catarrh and chronic intestinal catarrh, both of which taken conjointly may be termed chronic dyspepsia (indigestion), which is a common disease among horses. It is probable that some chronic catarrhs of the stomach and intestines are merely symptoms of other diseases, such as organic diseases of the stomach and intestines, and affections of the lungs, heart and kidneys. This is a diagnostic fact that deserves special attention in the case of old horses.

Etiology.—Some animals are specially susceptible to chronic catarrh. Continued mal-nutrition, anæmic and cachectic conditions such as those produced by severe illness, chronic poisoning, over exertion, etc. predispose an animal to chronic gastro-intestinal catarrh. Chronic catarrh often arises also from the acute form, when the dietetic causes which have been discussed under the heading of acute catarrh are continued or often repeated. The horses of millers frequently get this disease from almost exclusive feeding on bran and short-cut straw. Chronic catarrh of the stomach also arises from persistent wind-sucking.

Dental defects, by impairing mastication and salivation of food, are frequently the indirect cause in old animals of chronic inflammatory conditions of the bowels. The feeding of old and weakly animals upon uncrushed oats is a common cause of catarrh. It may also proceed from morbid dental conditions such as caries, alveolar periostitis, or the projection of single back teeth; and from the many abnormal formations (smooth,

jagged, zig-zag, ladder and scissor denture) which are included in the term dental anomalies.

Also, chronic catarrh is gradually developed by conditions which cause a permanent congestion of the mucous membrane of the stomach and intestines, such as frequent previous attacks of acute catarrh; congestion of the portal system, caused by chronic diseases of the liver, and more rarely by disease of the portal vein, congestion of the posterior vena cava in consequence of affections of the heart or lungs (valvular defects, pulmonary emphysema, and induration of the lungs), congestion of the intestine itself, especially circumscribed hyperæmia of the intestine by embolic obstruction of certain arteries in verminous aneurism. In all these cases the catarrh is not the immediate consequence of the congestion; but is rather a secondary affection, in that the intestinal mucous membrane suffers nutritive disturbances in consequence of the abnormal distribution of blood, and thus becomes less resistant to external irritants. Therefore, catarrh is often absent in such cases of venous congestion in the intestine. In this way chronic diarrhœa, brought on merely by frequent stimulation of the peristaltic movements of the intestines (as for instance, by chill), and the congestion consequent thereupon, occasionally set up chronic catarrh.

Sometimes chronic catarrh is due to the presence in the stomach and intestines of injurious entozoa, especially bots, which as a rule, however, are not hurtful, but sometimes, particularly when very numerous, produce considerable digestive disturbance, especially in foals. Besides *Gastrophilus equi* and *pecorum*, we have also to consider the larvæ of *Gastrophilus hæmorrhoidalis* and *nasalis* which are often found in great numbers in the small intestine. Chronic gastric catarrh is occasionally set up by *Filaria s. spiroptera megastoma*, which is usually met with in the right sac of the stomach in the form of submucous cysts, varying in size from a bean to an apple. Respecting true entozoa (*Ascaris megalocephala, tænia mamillana, plicata* and *perfoliata*) see the chapter on "Helminthiasis."

Symptomatic chronic gastro-intestinal catarrh is caused by various organic changes in the stomach and intestines; as for instance by ulcers, dilatations, stenoses, displacements, new-growths, foreign bodies (concretions and calculi), impaction of food, affections of the pancreas (*e.g.* colloid carcinoma) and new-growths of the liver. Chronic catarrh of

the stomach is a symptom of various chronic diseases, such as anæmia, rickets, and leucæmia.

Kumgata disease.—Under this name ("sand-disease") Vederniikoff has described a fatal disease which occurs among the horses of the Kirgis steppes, and which is accompanied by chronic gastric catarrh. On *post mortem* examination, large quantities (up to 10 to 12 lb.) of sand mixed with food are found in the stomach along with inflammatory changes of the mucous membrane.

Anatomical Conditions.—1. *Condition of the stomach.* The anatomical appearances vary according to the severity and duration of the complaint. In mild cases nothing important is noticeable. The changes are usually found in the mucous membrane of the right sac, and especially on the great curve, and in the neighbourhood of the pylorus. The mucous membrane is covered with a layer of ropy, glairy mucus, which may be of a light grey colour on account of the abundance of its contained epithelium, and which adheres firmly to it. The colour of the mucous membrane is by no means a reliable sign, and we must avoid mistaking normal irregular colouring of the right sac for pathological alterations. In chronic catarrh, the mucous membrane usually varies in colour from a dull red to a dirty red brown, and is sometimes slate-grey and spotted, so that it assumes a marbled appearance. The discoloration is due to the deposition of black pigment granules, which come from the hæmoglobin of extravasated red blood corpuscles, or from capillary hæmorrhages. In other cases we find the mucous membrane to be of a pale buff colour, and intersected by a few greatly dilated blood vessels. The surface of the mucous membrane is either even and smooth, or peculiarly uneven and rugged, corrugated, papillary and warty, or it forms stiff folds and ridges, and in rare cases, cone-like proliferations (gastritis polyposa—polyposis). Only in one instance have we observed microscopically a thinning (atrophy) of the mucous membrane ; but hypertrophy of the mucous membrane, which may become even five times as thick as it is normally, often occurs, especially in the warty condition, and in polyposis. The thickened tissue is, as a rule, compact, inelastic and fibrous. On being cut, it offers considerable resistance to the knife, and sometimes gives out a crackling sound, and the stiff edges of the incision do not retract. We can often see with our unaided eyes an increase of the submucous connective

tissue and a thickening of the muscles, which are also hard to cut. In other cases of chronic gastric catarrh, dilatation of the stomach is caused by relaxation and thinning of the muscles, when the stomach increases to double, or treble its normal size. The remarks on ulcers of the stomach (p. 8) may be consulted with respect to ulceration in this chronic catarrh. In very severe cases, the mucous membrane of the left sac may also become implicated.

2. *Condition of the intestine.* The morbid appearances in intestinal catarrh resemble those of the stomach. Here also we find that the mucous membrane sometimes has dark red streaks and spots, and at other times is of a slate-grey colour resembling that of an eel's skin ; the vessels appear very sinuous and dilated; and the surface of the mucous membrane is covered with mucus which is ropy, grey, glairy or purulent, and sometimes lumpy. The follicles are at first swollen, and later on become atrophied and disappear, when the mucous membrane assumes a latticed areolated appearance. The contents of the small intestine may become very watery, and often resemble whey clouded with flakes. Although the mucous membrane is on rare occasions thin, it is generally considerably thickened, and is very compact and wrinkled in consequence of the proliferation of the interglandular and submucous connective tissue. In the small intestine we frequently find papillary and polypoid proliferations similar to those in the stomach. Contractions and dilations in consequence of, respectively, hypertrophy and atrophy of the intestinal muscles, are likewise met with in chronic catarrh.

In the large intestine, and especially in stomach-like dilatations of the colon, the follicles frequently take a very active part in the inflammatory process, and either project in the form of nodular swellings, which are of a mucous, purulent, or thick pappy nature and vary in size from a grain of millet, to a pea; or they develop, by ulceration, into ulcers, which frequently cover a considerable portion of the large intestine. Röll states that they vary from the size of a hemp seed to that of a lentil; that they extend as far down as the submucous connective tissue; that they have sharp, non-infiltrated, undermined edges and a suppurating base; that they frequently form large ulcers by confluence; and that they either heal and leave behind a slight cicatrix, or cause perforation of the intestine, after having produced repeated diarrhœa and colic during a long period. (See "Intestinal Ulcers," p. 8.)

Symptoms.—1. *Symptoms of chronic gastric catarrh.* The principal symptoms of a catarrh which is chiefly confined to the stomach, are, as in acute catarrh, loss and capriciousness of appetite, frequent yawning, delayed defecation, coated tongue, dry and slimy mouth. There is as a rule no presence of fever. These symptoms, which are often the only ones visible at the beginning of an attack, usually undergo a remarkable change later on. The appetite is greatly disturbed after severe exertion, or even after ordinary work; but may soon become more or less normal. Even slight faults in feeding may aggravate the condition, especially by bringing on colic; and, consequently, frequent attacks of colic are by no means rare in chronic gastric catarrh. In severe and long protracted cases we sometimes find peculiar nervous derangements, which were formerly comprised under the name of "stomach staggers." Besides debility and disinclination for work, which are always present, the animal may exhibit cerebral depression and even vertigo. Attempts to explain these mental disturbances have been made by ascribing the cause to conduction of the nervous irritation of the stomach to the brain; anæmia of the brain produced by hyperæmia of the alimentary canal; and absorption into the blood of abnormal substances formed in the affected stomach (auto-infection).

2. *Symptoms of chronic intestinal catarrh.* The symptoms vary greatly according to the intensity, duration and extent of the complaint. The abdomen is usually empty and tucked up, although at times it may be tympanitic; the intestinal sounds vary greatly in their nature, but are generally more or less suppressed; and frequently constipation appears, the fæces being often formed into small balls, dry, with an acid or putrid smell, and sometimes remarkably pale and rich in undigested food. In other cases we may find the chief symptom to be chronic diarrhœa, which will then indicate the presence of chronic intestinal catarrh. Constipation and diarrhœa often occur alternately. We usually observe flatulence and frequently recurring signs of uneasiness. Long-standing cases are marked by mal-nutrition; the animal becomes weak, emaciated and sweats readily; the mucous membranes are pale or yellow; the hair is staring and lustreless; the pulse, small and weak, and the action of the heart irregular and often intermittent. The urine affords no certain indication, and in some cases is quite normal. As in acute intestinal catarrh, its colour varies greatly and it is frequently slimy. In most cases its

reaction is alkaline, which statement is confirmed by Albrecht; but Siedamgrotzky and Hofmeister report that they have almost invariably found it acid. Occasionally the urine gives a slight acid reaction, which may be attributed to exacerbations of the chronic process, or to a special affection of the small intestine. In a few cases we have found large quantities of albumen in the urine.

Duration and Course.—The duration depends on the possibility of removing the causes, and on the extent and intensity of the disease. The more easily the origin can be determined, the quicker can it be removed, and the shorter and more favourable will be the course of the malady. This remark is specially applicable to catarrh caused by faults in feeding. Severe lesions in the mucous membrane will, however, delay recovery. Horses which suffer from chronic catarrh of the stomach frequently continue at work for years, while constantly undergoing alternative improvement any exacerbation, although they gradually lose their strength and weight under such a condition. As a rule, such animals are brought to veterinary surgeons for treatment, not on account of the chronic disease, but in consequence of the recurring exacerbations. In other cases increasing weakness causes total incapacity for work, and death from exhaustion soon follows. It is probable that the majority of old and decrepit horses perish in this manner. If the chronic catarrh produces dilatation of the stomach, sudden death from colic may sometimes occur.

Treatment.—The principles of treatment are essentially the same as those for acute catarrh (p. 64). Attention should be chiefly paid to removal of the causes and to proper feeding. All indigestible, spoilt or unwholesome food should be avoided, and we should give digestible food in small and frequent quantities. For instance, we may add an extra meal in the forenoon and afternoon, and regulate the diet according to the work. In some cases, the addition of molasses to the food has been attended by good results. It has also a certain effect in preventing colic. Dental defects should be treated surgically, and particularly should any sharp points of teeth be removed, whilst the oats given to old animals should be bruised. Owners have often extreme difficulty in establishing a proper method of feeding in their stables.

Medical treatment is only of secondary importance. Hydro-

chloric acid in small doses (2 to 3 drams), has also proved itself here to be very efficacious. The principal part in the therapeutics of this disease is, however, played by alkalies, as is acknowledged by all practitioners. For instance, we may give a tablespoonful of common salt in each feed for some time, and bicarbonate of soda and Glauber's salts in doses of ¾ oz. to 1½ oz. The simultaneous administration of the above-mentioned three salts in the form of artificial Carlsbad salt is particularly efficacious. Carlsbad salt in doses of ¾ oz. to 1½ oz. neutralises acidity; stimulates, by its contained salt, the secretion of the digestive fluids; prevents fermentation; dissolves ropy mucus, and by its contained sulphate of soda causes evacuation of the fæces. As in acute catarrh of the stomach, we may also use rhubarb (a very valuable drug), calamus, gentian, aloes, ginger, anise, fennel, cumin, etc. Pepsin (1¾ to 2½ drs.), apomorphine (¾ to 3 grs.), and subnitrate of bismuth (½ to 1¾ drs.), may also be tried. For constipation we may employ green food and mashes; comparatively large doses of Glauber's salts (½ to 1 lb.); medium doses of aloes; and enemas of cold water, solutions of sodium chloride, soap and water, glycerine, etc. Kattner, Schindelka and others recommend the injection of glycerine (3 to 6 oz.) into the rectum by means of a syringe having a long tube. It has a very irritating effect on the mucous membrane of the large intestine and produces peristalsis and defecation by reflex action. We have found that enemas of glycerine have about the same effect as those of soap and water, the action of both being simply to clear out the rectum. They have no power to remove fæcal accumulations which are situated higher up in the intestine, and are consequently useless in colic caused by impaction of food.

For diarrhœa we may use the astringent and styptic remedies recommended for acute catarrh, oak-bark, cinchona bark, tannin, tannoform, tannalbin, alum, ferrous sulphate, acetate of lead, subnitrate of bismuth, nitrate of silver. These remedies should, however, be used with caution, as after repeated employment they are liable to lose their efficacy. Opium is in all cases the most reliable agent. In chronic catarrh of the rectum styptic agents are best given in the form of an enema, but it must be remembered that such injections have no effect whatsoever upon the mucous membrane of the colon or cæcum. According to Albrecht, creolin (2 to 3 drams daily) is the best remedy in chronic gastro-intestinal catarrh. By its action the appetite and digestion quickly improve, peristalsis becomes

stimulated, and the fæces almost immediately lose their bad odour. Kramell confirms this view of the favourable action of creolin in chronic diarrhœa.

ACUTE GASTRO-INTESTINAL CATARRH OF CATTLE.

(*Acute bovine dyspepsia.*)

General Observations upon the Diseases of the Gastro-intestinal Canal in Ruminants.—It is an undeniable fact that, in spite of the importance to both veterinary surgeon and agriculturalist of the diseases which affect the digestive organs of ruminants, our knowledge of the subject is very incomplete. In the absence of any certain foundations for differential diagnosis, pathologists have contented themselves with grouping together under the general heading of "Indigestion" a number of very varied morbid conditions of the gastro-intestinal canal. Latterly, however, special efforts have been directed towards the classification of these affections according to their character and locality; to the presentation of well-defined descriptions of the clinical aspects of each condition, and especially to the establishment of so-called pathognomonic symptoms as aids to diagnosis. Such efforts deserve every recognition and it is to be hoped that further help of the kind may be forthcoming. It must not be forgotten, however, that the difficulty of forming an exact diagnosis is far greater in the case of the ruminants than in that of the dog or the horse. In order to realise this, it is only necessary to consider for a moment the complicated character of the stomach of ruminants, with its four divisions, every one differing both in structure and function from the other three, yet each dependent upon the others and influenced by the conditions which affect them. It frequently happens that a pathological condition affecting one portion will involve the other three, and thus, in a certain sense, there may be several morbid conditions existing at one and the same time. For this reason, it is extremely difficult to exactly classify the different gastric disorders of ruminants and, in the present state of our knowledge, it is practically impossible to give an exact and clearly defined clinical description of them. We may, indeed, go so far as to say that a differential diagnosis of these conditions, in spite of the closest investigation, is bound to be imperfect. In the earlier stages of disease diagnosis is frequently impossible,

the earlier symptoms being, in most cases, remarkably similar and anything but pathognomonic in character.

In this chapter, we have endeavoured to arrange the subject matter in such a way, as first to deal with acute and chronic gastro-intestinal catarrh ; then with acute and chronic tympanites and impaction of the rumen ; followed by catarrh of the abomasum ; gastro-intestinal catarrh in young cattle ; and finally the disturbances of digestion brought about by the presence of foreign bodies in the paunch or reticulum. This arrangement should prove of considerable practical value.

With cattle, as with horses, we have preferred to discuss so-called acute dyspepsia along with acute gastro-intestinal catarrh, and not as an independent disease. It appears that in the beginning of many bovine cases the alteration is in the function and not in the structure of the implicated organ. This is the more probable, because the mucous membrane of the first three stomachs is very resistant to a catarrhal affection. For instance, rumination may be entirely suppressed merely on account of nervous or motor disturbances of the rumen, without any previous inflammation of its mucous membrane. As these anomalies of function, when other than quite transitory, are directly followed by acute catarrh (the causes of dyspepsia and catarrh being the same), and as their respective differentiating symptoms cannot be distinguished from each other, we have thought it best, contrary to usual custom, to describe acute dyspepsia only as the first stage of acute gastro-intestinal catarrh.

In conformity with the complicated structure of the stomach of ruminants, we find that the three ante-stomachs are chiefly attacked, and especially the rumen and omasum. In some cases the abomasum and duodenum are prominently implicated. Usually the intestine is involved only sympathetically, and its affection offers, externally, few characteristic symptoms, although sometimes even the affection of the intestine may become very prominent, in which case diarrhœa will form the chief symptom.

Etiology.—The disposition to acute gastro-intestinal catarrh varies with individuals. This is illustrated by the fact that in a whole byre of cattle, all receiving the same food, single animals *only* will develop the disease. The origin of these isolated cases is not very clear, but frequently they can be attributed to weakness of digestion, relaxed constitution, pre-

vious disease, age, etc. The principal pre-disposing causes are errors of feeding, such as the practice of giving relaxing food without sufficient rough fodder; keeping the animals constantly stabled; advanced gestation; or forcing the milk and, later on, fattening quickly. In small byres, belonging to poor people, especially near towns and in times of scarcity, animals are often fed on any collected refuse. In consequence of this unnatural method of feeding and housing, cows and goats are affected more readily than others and, for the same reason, the condition is to be met with much more frequently in certain districts and upon certain farms than others. The disease is more rife during spring and autumn than at other times. The following are the chief direct causes :—

1. Overfeeding.

2. Fodder which is frozen or covered with hoar frost; pasturing too early in the spring and too late in autumn; unaccustomed cold drinks; chills and too hot food.

3. Improper feeding, such as abrupt changes from dry to green food, and *vice versa*; too new hay, especially if the animals soon afterwards drink large quantities of water; excessive feeding on straw, bran, pollard, roots, potatoes, oilcake, unbruised grain, chaff, bye-products from breweries and distilleries; admixture of weeds, and turnip tops; sudden changes to unaccustomed food; fattening on dry food, etc. Although feeding on short-cut straw is generally regarded as injurious, the experiments made by Friedberger do not unconditionally confirm that idea.

4. Spoilt and decomposing food, such as kitchen refuse; fermenting and acid refuse from factories; hay which has been exposed to inundations, etc.

5. Indigestible food, such as tough, lignified plants; green flax; the eating of the after-birth by cows; and feeding on half-baked bread.

6. Foreign bodies of every kind, especially large quantities of earth, after feeding on roots; sand; mud and gravel.

7. Too severe and too long continued exertion, which causes rumination to cease, such as draught work or difficult parturition. We may point out that hard though well regulated draught-work, which the oxen of millers, brewers, farmers and others on the Continent have to do, causes no disturbance of digestion.

Symptoms.—1. *The dyspeptic stage of acute gastro-intestinal catarrh.* The initial symptoms of this disease are frequently

restricted to disturbances of appetite and digestion, when the animal loses its appetite, prefers green stuff to dry fodder, and fresh spring water to its usual drink, or it may begin to feed greedily and then soon stop eating ; it lies down a good deal, becomes depressed, and ceases to lick out its nostrils with its tongue ; rumination becomes infrequent and shortened in duration ; the movements of the paunch suppressed or slow ; defecation is somewhat delayed, and the fæces are normal or inclined to be dry. There is no fever, the extremities are warm, the muzzle is cool and moist, and the secretion of milk but little diminished. If the morbid condition does not pass off in, say, four to five days, it will develop into true catarrh.

2. *True acute catarrh of the stomach and intestines* (which either commences as such, or develops from the condition described in the preceding paragraph). It would be difficult to sketch a typical picture of the disease, and we shall therefore content ourselves by briefly noting the usual symptoms, which are as follows :—The animal is depressed and stands back almost constantly from the feeding trough ; he arches his back and places his feet under the body (like a chamois) ; the coat stands on end, especially on the head, over the eyebrows, and along the spine ; the ears hang down or lie back ; the temperature of the surface varies, particularly that of the ears and base of the horns, and occasionally there are slight rigors. The temperature of the muzzle is also variable; it still remains moist, though to a less extent than in its normal condition, and if the beads of moisture on it be wiped off, a considerable space of time will pass before they are replaced. Usually the conjunctiva is highly injected.

The oral cavity is abnormally warm ; the mucus and saliva contained in it are increased in amount, and even salivation is sometimes observed ; appetite and thirst are greatly diminished or entirely suppressed, and rumination generally ceases ; even if it continue to exist, it proceeds slowly and in a languid manner ; the morsels are small, and each bolus receives only about half of the usual number of grinding movements ; we may occasionally notice eructations of fœtid gases and even vomiting. As a rule the abdomen is distended, particularly on the left side, both above and below, and the left flank abnormally prominent. The contents of the paunch may be felt at the flank as a consistent dough-like mass, but if the wall of the flank is puffed out with the gases which have collected

in the upper portion of the abdomen, we can feel the semi-solid contents only by energetic palpation, in which case a dull knocking sound is heard. Usually there is a temporary change from time to time in the fulness of the left flank, in consequence of the oft recurring inflation. The movements and sounds of the rumen are reduced, although its contents are often heaved up, but they are not rotated as much as during health. Pressure on the left flank and hypochondrium does not appear to be painful.

Defecation is usually retarded. The dung is generally more solid and darker in colour than normal—annulated, sometimes fœtid, and may be covered with a thin crust, or it may be shining, pasty and surrounded with mucus. If watery diarrhœa comes on, we shall often find in it particles of entirely undigested food. By auscultation of the right wall of the abdomen, we may hear rumbling and rattling intestinal murmurs. Besides these symptoms, the animal is often somewhat restless, knuckles over with the hind feet, kicks at its belly, whisks its tail, repeatedly lies down and gets up, looks round at its hind quarters, assumes a lateral recumbent position, etc. In other cases severe constipation may ensue. The urine, which at the beginning of the attack was alkaline and of a bright sherry-yellow colour, becomes scanty later on, dark in colour and acid. The milk often decreases rapidly down to as little as half its usual quantity. Occasionally a pimply rash appears which, however, quickly passes off.

Fever is generally present. The pulse, as a rule, is small, hard and accelerated by from 10 to 20 beats per minute. The temperature of the rectum is increased, though not always in a uniform manner, on which account, as Harms has justly pointed out, we cannot always judge with certainty the severity of the malady from the state of the internal temperature. Moreover, we should bear in mind that in cattle the pulse and temperature vary a good deal, even under normal conditions. We shall, therefore, do well to feel the pulse and take the temperature of some of the healthy animals of the same herd or byre.

Process and Termination.—In mild cases, which are the rule, improvement begins in from five to eight days, the movements of the paunch become more frequent, and its sounds more distinct; rumination and appetite gradually reappear; defecation becomes more normal; the secretion of milk increases,

and fever gradually abates. In severe cases, on the contrary, the symptoms become intensified; the mouth is dry, coated, hot and emits a bad smell; rumination, consumption of food and the movements of the paunch are suppressed; either constipation or diarrhœa is present; milk secretion is arrested and the udder is flabby and relaxed; the movements are feeble, the sensory apparatus depressed, and the animal utters faint groans and sighs when lying down. The expression is plaintive; the eyes are sunken; mucus resembling sago accumulates at the inner angle of the eye; the fever rises to 40° C. (104° F.), or more, and the rate of the pulse is increased by 30 or 40 beats a minute. The ears and horns are alternatively hot and cold; the muzzle remains continually hot and dry; and respiration, which was quick from the beginning, becomes painful and still more accelerated. With these symptoms the disease approaches in character inflammation of the stomach and intestines, which usually ends in death. If acute catarrh runs a long course, it may pass into the chronic form.

Differential Diagnosis.—The symptoms, duration, course and termination of acute gastro-intestinal catarrh of cattle are, on the whole, so characteristic, that its diagnosis is easy. It is often impossible to determine which part of the alimentary canal is chiefly affected; but this is not of much importance, because, as a rule, the affection spreads rapidly from its first site to the neighbouring parts. The accumulation of gas in the rumen might cause acute catarrh to be mistaken for tympanites; but the slight amount of the flatulence, and its periodic and varying nature furnish ample data for differentiation. It is often very difficult to distinguish severe acute catarrh from inflammation of the stomach and intestines, especially as one may develop into the other. Owing to the accumulation of small quantities of gas in the paunch, acute catarrh may sometimes be mistaken for tympanites. But the formation of wind is very slight, while the periodic nature of the disease and its varying course furnish ample grounds for differentiation. In cases where the symptoms are very severe, there is a certain danger of confusing simple gastric catarrh with inflammation of the bowels. It is all the more difficult to distinguish between the two conditions as one frequently results from the other. In cattle, too, torsio uteri may for a time be confounded with indigestion, and for this reason (according to Wolf and Albrecht) cows

According to Schneider, gastric disturbances during the later stages of pregnancy are frequently mistaken by the uninitiated for labour pains, and the extreme measures which are adopted in such cases often result in the bringing on of true labour pains.

Prognosis.—Recovery is nearly certain in simple catarrh. Hering states that in 128 cases of "indigestion," only five were slaughtered, and that resort to this measure was not absolutely necessary. Acute catarrh may, however, develop into chronic catarrh, which is more dangerous to life than the acute form, and may even run into inflammation. Consequently, we should make our prognosis at the beginning of the attack with a certain amount of reserve.

Post-Mortem Conditions.—Owing to the fact that very few *post-mortem* examinations are made on account of the small percentage of deaths, our knowledge of the pathological anatomy of the disease is small. Harms states that the mucous membrane is generally swollen, congested in stripes, in spots, or diffusely, and that, especially in the abomasum and intestine, it is covered with a coating rich in cells. The catarrhal symptoms are best marked on those parts of the rumen which possess large papillæ, and are less evident in the reticulum and omasum, of which a few leaves are now and then severely affected. Some authorities state that the contents of the omasum are in places quite dry, and of a coarse hard nature in the vicinity of the free edges of the large laminæ.

Treatment.—The treatment of acute gastro-intestinal catarrh in cattle consists: (1) in the removal of the originating causes; and (2) in a strict diet. So long as rumination is suppressed, we should keep the animal more or less fasting, and give it nothing except a very little easily digestible food, as, for instance, freshly-mown meadow grass, tender hay or aftermath, more with a view to stimulate the movements of the stomach, than to satisfy any appetite which might be present. The animal should be kept on short diet, even after rumination has become re-established, because active treatment cannot be applied until the rumen is freed from the masses of food which have accumulated in it. At the same time we should encourage thirst by giving salted gruel or whey. If these are refused, we may gain our object by getting the patient frequently to lick

salt. The movements of the stomach and intestines may be stimulated by hand-rubbing the surface of the trunk, and especially over the region of the paunch from the left flank. If defecation be delayed, the animal should be frequently back-raked, and enemas of soap and water or salt and water employed.

If simple dyspeptic symptoms exist, we may give 2 or 3 drams of hydrochloric acid internally two or three times daily in a bottle of water. If at the same time there is constipation, as is usually the case, the milder purgatives may be employed, such as sulphate of magnesia or Glauber's salts, in combination with mucilaginous food or with infusions of linseed, althœa-root, or fenugreek. Glauber's salts (1½ lbs.) for instance, may be dissolved in two or three quarts of linseed tea, and divided into three doses to be given at intervals of about three hours, or the salts may be dissolved in an infusion of bitters (gentian) or one of the bitter aromatics (calamus). For retarded or suppressed rumination tartar emetic and hellebore are the best stimulants, either of which may be given in doses of 2 to 4 drams; or we may inject subcutaneously 1½ to 2 grains of veratrine, dissolved in ¼ oz. of 2% alcohol. Some advise creolin in doses of from ½ to 1 oz. Sulphate of physostigmine has been employed with good results for atony and paralysis of the stomach and intestine. Feser, as the outcome of his experiments with cattle of the smaller breeds, recommends 1½ grains of it dissolved in 40 minims of water, to be injected under the skin; which may be repeated in five or six hours if the desired result has not been attained. Eber considers that the best agent is eseridinum tartaricum (2¼ to 4½ grains), and barium chloride has been recommended. Pilocarpinum muriaticum has also been suggested, but it is not so efficacious with cattle as physostigmine and, owing to its sialagogic action, is attended by undesirable effects on the salivary glands. This is also the case when physostigmine is used in combination with pilocarpine; it is also so with arecoline. Under certain conditions, puncture of the rumen and the use of a probang may be necessary. Where diarrhœa is present, tannoform, tannalbin, and tannopin have latterly been recommended in place of tannin. According to Schindelka repeated doses of tannopin of 2½ drams each have a prompt effect in reducing diarrhœa.

ACUTE GASTRO-INTESTINAL CATARRH OF SHEEP.

In this disease we find the same etiological and symptomatic conditions as in the bovine form. Chill and errors of feeding are their chief causes. Gerlach observed gastro-intestinal catarrh in flocks of sheep which had been pastured on luxuriant meadows; and Schick had a similar experience with sheep that had been fed on large quantities of raw potatoes. Here also the chief symptoms are: diminished consumption of food; disturbances of rumination; thin and even watery diarrhœa; occasional constipation; faintness, etc. As compared with cattle, sheep are much more liable to succumb to this disease, in consequence of their weaker constitution. Schick reports that in a herd of 600 sheep, 60 died within a week; and Gerlach, that in a herd of 300 sheep, 20 were dead in four days. On *post-mortem* examination, the changes were found to be similar to those produced "by severe cathartics." Owing to the fact that the disease often attacks whole herds of cattle, it is sometimes looked upon as infectious.

CHRONIC GASTRO-INTESTINAL CATARRH IN CATTLE.

(*Chronic dyspepsia or Omasitis chronica.*)

General Remarks.—Chronic gastro-intestinal catarrh of cattle is generally described under the name of "impaction of the omasum." This designation is due to the fact that the accumulation of firm, dry masses of food in the omasum was always considered to be the chief morbid aspect of the disease, which was never diagnosed as a chronic catarrh of the stomach or intestine, as in other animals, but always as an omasitis, namely, so-called impaction of the omasum. This name is by no means distinctive, as has been pointed out for a long time by many observers. In the first place, such an accumulation of food in the omasum occurs in various bovine maladies, during the course of which, appetite, rumination and the movements of the paunch are for a long time suppressed, and the contents of the omasum become gradually dry from absence of saliva, or from absorption of the fluid. The condition in question is met with in all severe febrile diseases, and particularly in digestive troubles. For instance, rinderpest was formerly called "dry omasum." Furthermore, a true obstruction to the passage of food through the omasum hardly ever occurs. Harms has

observed only one case. The old name should therefore be given up.

There are, however, cases in which the catarrh may become prominently localised in the omasum, but they are rarer than the chronic affections of the paunch, abomasum and intestine. Catarrh of the omasum is generally symptomatic of a disease of the rumen, etc. As it is impossible to give a separate description of the respective catarrhs of the intestine and of each of the four stomachs, we have comprised the chronic inflammatory condition of the mucous membrane of these divisions of the alimentary canal under one heading, namely, "chronic gastro-intestinal catarrh."

Etiology.—All the causes of acute catarrh described may produce chronic catarrh, provided that they are more continuous or more intense. Neglected or irrational treatment of acute catarrh frequently induces chronic catarrh. We have also to consider the following factors :—

1. Imperfect mastication, owing to defective teeth or absence of teeth.

2. Adhesion of the rumen or reticulum to the abdominal wall, in consequence of circumscribed peritonitis.

3. Compression of the œsophagus by new growths, and consequent impediment to rumination.

4. New growths in the various compartments of the stomach, as, for instance, scirrhus and sarcomata in the abomasum; papillary proliferations at the opening of the reticulum and omasum; and papillomata and armour-like sarcomata, etc.

5. Displacement of the reticulum by rupture of the diaphragm; diaphragmatic hernia of the reticulum.

6. Foreign bodies in the stomach and intestines, such as nails, and balls of hair, vegetable matter or wool; old, musty straw; sand, mud and dirt, due to the eating of fodder from flooded meadows; new growths; diverticula dilatations and stenoses of the paunch and intestines; multiple polypous proliferations in the small intestine; and intestinal vesicular fistulæ.

7. Vascular engorgement in chronic diseases of the liver, lungs and heart.

8. Pressure of the gravid uterus on the rumen and weakness of the movements of the rumen.

9. Chronic gastro-intestinal catarrh is found secondarily in all chronic disturbances of nutrition, especially in tuberculosis.

Symptoms.—The symptoms of chronic catarrh differ but little from the acute form, except as regards their longer duration and greater persistency. The disease is characterised by frequent remissions and exacerbations. The general health is upset; the animal is depressed and feverish; the temperature of the body is unequally distributed; the skin hidebound; the coat rough and lustreless; gradually increasing emaciation appears; the mouth shows changes similar to those described in acute catarrh; consumption of food, rumination and the movements of the stomach are diminished or entirely stopped; signs of pica (p. 42) are sometimes present; eructations are rare, but when they occur, are fœtid; the paunch is usually hard, firm to the touch and considerably filled with food; when the flanks are sharply tapped a distinct knocking sound is heard. We can generally observe slight inflation of the rumen, which varies according to the consumption of food, and becomes continuous after the movement of the stomach has ceased. Vomiting has been noticed in a few cases.

Defecation is considerably delayed, and the scanty dung is passed in the form of dry, peat-like, dark-coloured balls, which often contain many particles of imperfectly masticated and undigested food, and are generally covered with tenacious, glairy or bloody mucus, sometimes in shreds. At other times soft, thin, acid or stinking masses are passed, in which case the diarrhœa will last for weeks, if not months, and may lead to death from exhaustion. Usually constipation and diarrhœa alternate. The quantity of urine is generally small, and is proportionate to the decreased consumption of water. We have no exact statements as to its reaction. Milk secretion is diminished or entirely arrested. Groaning and grinding of the teeth are often observed.

If recovery does not set in, the intensity of the symptoms, as a rule, will quickly increase. The appetite is lost; the muzzle becomes dry and fissured; the fever higher; the pulse small and weak; the heart palpitating; tympany increases; the movements of the stomach are entirely suspended; complete constipation or persistent diarrhœa sets in, and emaciation increases. The animal is very weak and prostrate; lies down a great deal, groans uninterruptedly, and is eventually incapable of getting up. The expression is plaintive, the eyes sunken, and death takes place, either naturally, or by slaughter.

Post-Mortem Conditions.—The anatomical changes

vary greatly, according to the localisation, intensity and duration of the chronic catarrh.

The *rumen* is somewhat inflated by gas, and contains a great quantity of dry food. The epithelium of the mucous membrane can often be readily detached soon after death, although in a healthy carcase it remains firmly adherent for a long time. According to Bruchmüller, it is occasionally thickened. The mucous membrane, especially in the part which has large papillæ, is interspersed with ecchymoses, either uniformly or in spots, and is occasionally swollen, thickened and of a purple or brown-red colour. The papillæ may be either in an atrophied or hypertrophied condition.

The *reticulum* is usually least affected, and sometimes contains a large quantity of dried-up coarse food. Its mucous membrane has appearances similar to those in the rumen.

The *omasum* is considerably dilated and completely filled with food, the cakes between its leaves being compressed and unusually dry. The epithelium of the mucous membrane is also abnormally detachable, and often adheres to the dry cakes. The ingesta is either coarser than in the rumen, or is finer, because it has been ruminated, and can be broken up between the fingers into a fine granular powder. The mucous membrane of the leaves may be congested diffusely, in spots, or in streaks; may be inflamed, soft and tender; gangrenous, or interspersed with dark-red foci and hæmorrhagic ulcers.

The *abomasum*, as a rule, is more or less empty, and often contains coarse chyme. Its mucous membrane shows an intense infiltration of pigment in the form of slate-grey streaks and spots. It is also thin and smooth (atrophied) and covered with a tenacious, glairy fluid.

The *intestinal canal* shows various changes. Its mucous membrane may be markedly congested, changed in colour to slate-grey, atrophied, and cribriform and pitted, in consequence of the obliteration of the Peyer's patches. The contents of the *duodenum* usually consist of large quantities of bile-coloured mucous masses. In the *large intestine* we generally find dry, dark coloured fæces, and the mucous membrane covered with a thick coating. When chronic catarrh turns into phlegmonous inflammation of the intestine, or into peritonitis, we have the respective changes of the two last-mentioned diseases. The *gall-bladder* is generally filled with thick bile. The *liver* shows various stages of engorgement.

The other conditions vary greatly, and may usually be

referred to venous congestion. They consist of hyperæmia of the lungs, meninges of the brain, kidneys, right side of the heart, venous system, etc.

Differential Diagnosis.—Owing to its very varied aspect, chronic gastro-intestinal catarrh may be confused with several diseases, such as acute catarrh and acute phlegmonous inflammation of the intestines; but its chronic course will be its characteristic sign in both these cases. It may often be very difficult to distinguish it from chronic peritonitis, brought on perhaps by a perforating foreign body; because this peritonitis frequently becomes complicated with chronic catarrh, and the peritoneum may become sympathetically affected by an intense catarrh. In many cases we obtain an explanation of the nature of the malady only by the course of the disease (as by supervening symptoms of heart disease), or *post mortem*. A large accumulation of gas in the paunch being the chief symptom of acute or chronic tympanites, may suggest either of these diseases; because meteorism occurs but occasionally and only to a slight degree in chronic catarrh. Finally, chronic gastro-intestinal catarrh may be confounded with tuberculosis, which is a chronic disease that shows emaciation, fever, disturbances of digestion and nutrition, etc. An examination of the lungs, and the use of tuberculin will enable us to make a prompt and exact diagnosis.

Prognosis.—In chronic gastro-intestinal catarrh of cattle the prognosis is based (1) upon the nature of the originating causes and the possibility of their removal; and (2) upon the nature, extent and duration of the catarrhal condition itself. It is well to exercise caution in making a favourable prognosis in the beginning of the attack. There is small chance of recovery if the disease lasts longer than two or three weeks; if complete constipation be present for several days; if diarrhœa persists for a week or longer; or when the appetite, rumination and the movements of the intestine remain entirely suppressed. Slaughter will then be generally preferable to treatment.

Treatment.—The number of remedies used in chronic gastro-intestinal catarrh of cattle, and enjoying a certain reputation is considerable. We shall confine ourselves to a description of certain only of these, as it is our intention to limit as far as possible the list of drugs, here as elsewhere in this

book, to those which are really essential. Speaking generally, the therapeutic measures which have been described in connection with acute gastro-intestinal catarrh are equally applicable to it in its chronic form. Thus, slight cases, dyspeptic in character, should be strictly dieted and treated with hydrochloric acid in doses of 2 drams several times a day. Small doses of bicarbonate of soda, Glauber's salts, Carlsbad salts (1½ to 4½ oz.) given continuously over a period of several weeks have sometimes had excellent results. Where there is pronounced relaxation of the ante-stomach, provided always that actual symptoms of inflammation have not made their appearance, oil of turpentine (2½ to 6 drs. two or three times a day) in an aromatic infusion ; spirits of wine (¾ to 1½ oz. to a dose) ; and hellebore, either as pulv. rhizom. veratri or in the form of a tincture (2½ to 5 drs.), are valuable drugs. Subcutaneous injections of veratrine (¾ to 3 grs.) produce good results. Latterly creolin has been credited with excellent effects in chronic gastro-intestinal catarrh of cattle. Truelsen finds that creolin (1 to 2 drams in a bottle of water, three times daily) is the best remedy, and that, with it, recovery takes place in a few days. As aperients we can recommend large doses of Glauber's salts (16 to 32 ozs.), or Carlsbad salts (p. 72) with large quantities of water ; castor oil (1 to 2 pints) ; aloes (1 to 2 ozs.) ; and in cases of very obstinate constipation, tartar emetic (2½ to 5 dr.) or calomel (15 to 75 grains *pro die*), which should be used cautiously. The much-lauded sulphate of eserine (1½ gr. subcutaneously) appears by recent observations to be occasionally ineffective, and sometimes to cause abortion, dangerous dyspepsia, suffocation, etc. As a substitute for eserine, Eber recommends sulphate of eserinum tartaricum (¼ to ½ gr.). Wolff finds barium chloride effective. Croton oil, on account of its irritant and poisonous properties, should be used sparingly ; it should be replaced as far as possible by castor-oil (1 to 2 pints). In the treatment of bovine affections it should always be borne in mind that the animal may, eventually, have to be slaughtered, and care should be taken to adopt a line of treatment which is not likely to injure the meat. In addition to medicinal measures, the introduction of large quantities of water into the stomach and intestines is frequently attended by good results, and in obstinate cases, is often the only effective measure. Höhne and others have obtained many recoveries by filling the rumen with large quantities of warm water, or salt and water, by means of a trocar and funnel. Deffke injects

eleven gallons of luke-warm water in the course of several hours, the canula remaining in the whole time. He describes the excellent effects obtained by the addition of hydrochloric acid to the injections (2½ drs. to 3¼ gallons of water), as well as by the addition of infusions of certain aromatics, such as valerian root. He advises the adoption of this measure early in the course of the disease. Dreymann advocates the injection into the rectum of nine to thirteen gallons of warm water by means of a special irrigator. Raw linseed oil, mucilaginous infusions, and frequent irrigation of the rectum with water, are all advised.

If the rumen be filled with very firm masses of food, and medicines have proved useless, and there is a probability of foreign bodies being present in the stomach, we may open the rumen as a last resource, and remove a part of the contents.

Chronic Gastro-intestinal Catarrh in Buffaloes.—In 1898–9 Marcone observed an enzootic disease among buffalo calves after eating fodder with much sand in it off flooded meadows at Voturno, near Capua. The animals became emaciated, showed loss of appetite and suppressed rumination, sensitiveness of the flanks to pressure, and a grey heavy dung was passed consisting almost entirely of sand. Thirty-one out of eighty-five died. Müller writes of an equally high rate of mortality among cattle on the lands liable to inundation by the Elbe and Havel, and due to the eating of muddied grass and hay. Prietsch relates an instance where the fatal results were due to the hay becoming covered with a fine dust which drifted through the cracks in the hay-shed from a neighbouring stone-quarry where a stone-breaking machine was in use.

ACUTE TYMPANITES OF RUMINANTS.

Nature.—The clinical aspect of acute tympanites in ruminants differs considerably from that of the chronic form. The former, known generally as "tympanites," is a condition brought about by the sudden formation of an excessive quantity of gas in the intestine, and its accumulation in the paunch, giving rise to considerable inflation of the ante-stomach and consequent distension of the flanks. Its occurrence is most frequent among cattle and then amongst sheep. It may attack several animals at once and this is notably the case in flocks of sheep.

Etiology.—*General causes.* The chief cause is the introduction into the stomach of a large amount of food which quickly ferments and produces great quantities of gases. With stalled animals a fruitful cause is the ravenous feeding on stale or withered green-fodder, and especially upon green-stuff heated through being allowed to lie in heaps and thus fermenting. This fact explains the usual occurrence of this malady on holidays, particularly when there is a succession of these days, and a large supply of green food has been laid in. We also know from experience that this affection is most frequent during damp, warm weather, which stimulates fermentation. With pasture-fed animals the less they are accustomed to grazing and green food, and the richer the herbage upon which they feed, the more frequent will be the occurrence of tympanites among them. Experience has shown that the condition is more prevalent in those years when, after a cold spring, warmer weather sets in, and as a consequence of the sudden change, plant-life develops with unusual luxuriance (Spinola). It is also found to occur with greater frequency on low lands where the soil is heavy and fertile and the pasturage over-rich. Tympanites is very likely to follow if the animals are turned out to graze on meadows where there is a heavy dew, or hoar frost. or snow, or where the grass is soaked with rain ; if they are allowed to drink too soon after feeding ; if when at grass they take in an excessive amount of wind ; and if, owing to carelessness, they are allowed to stray into clover-fields, etc.

Undoubtedly certain animals have a pre-disposition to tympanites. Apart from the fact that some eat too quickly or too much, and so develop flatulence. There are others in whom a comparatively small quantity of fermenting food will provoke tympanites. This especially occurs with animals suffering from a weakness of the digestive organs, which is due to a relaxation of the muscles of the paunch, or to special pathological conditions affecting the paunch, or eructation. The last-named leads to a state of chronic flatulency.

Special causes.—In addition to the general causes enumerated above, there are certain special ones, such as the effects of various food plants, which must be noted when considering the origin of tympanites, such are :—

1. Clovers, such as red clover (*trifolium pratense*), lucerne (*medicago sativa*), sainfoin (*onobrychis sativa*). Clover is much more dangerous before blossoming than after it has become mature, and young red clover is especially harmful. Clover

grown on land manured with gypsum is said to be a cause of tympanites, but this is denied by Rinquet and others. In view of the fact, however, that gypsum promotes rank growth, the supposition appears feasible. The injurious effects of young clover are increased by the action of frost and snow (Honeker).

2. Other leguminous plants, such as green lentils or vetches and buckwheat.

3. Grasses grown on wet soil; reed grasses covered with microbes, such as *poa aquatica* and *scirpus sylvaticus*; potato and turnip tops; leaves of cabbage and rape; charlock (*sinapis arvensis*) and wild radish (*raphanus raphanistrum*).

4. Green corn; feeding on stubbles containing blades of corn and weeds. Plants grown in the dark are more dangerous than when they have been exposed for some time to sunlight. Feeding on stubble where much grain has fallen and sprouted should especially be avoided.

5. Fermenting drinks, brewers' by-products, skins of pressed grapes, distillers' wash, dried brewers' grains, yeast, rotten potatoes, turnip-tops and bulbous plants, cotton-seed meal, especially when the animal has not been accustomed to them, and when they are eaten hastily and in large quantities.

6. Tympanites may occur as a symptom of various poisonings, such as by meadow saffron (*colchicum autumnale*), hemlock (*conium maculatum*), water hemlock (*cicuta virosa*), deadly nightshade (*atropa belladonna*), yew (*taxus baccata*), white hellebore (*veratrum album*), common red poppy (*papaver rhoeas*), tobacco, various kinds of ranunculus, mould fungi, salt fish and salt-meat brine, etc.

7. Cattle sometimes blow themselves out in a manner analogous to the wind-sucking of horses. Calves also, at times, swallow an inordinate quantity of air when sucking the teat or other object.

8. Where the work is excessive, owing to suppressed eructation, tympanites may be produced under normal conditions of feeding.

9. Foreign bodies which have stuck in the œsophagus may produce tympanites by impeding eructation. We met with a case of this kind which was caused by a hairball that had been thrown up into the gullet from the stomach.

Post-Mortem Conditions.—The contents of the strongly distended paunch besides the food consist chiefly of the following gases: carbonic acid, carburetted hydrogen, hydrogen, sulphuretted hydrogen, oxygen and nitrogen. Carbonic acid

is in abundance when the animal has been fed on green food, and carburetted hydrogen when the forage has been dry. Reiset found that the contents of the rumen of a cow which had died from tympanites two hours after feeding in a field of clover was as follows :

Carbonic acid	74	per cent.
Carburetted hydrogen ..	24	„ „
Nitrogen	2	„ „

In a tympanitic wether, Reiset found 76 per cent. of carbonic acid, but no sulphuretted hydrogen, the absence of which appears to be accidental, because it may be a physiological product of the normal decomposition of albumen in the rumen.

The anatomical changes in acute tympanites consist, in the first place, of a pronounced distension of the paunch, which may lead to rupture both of the paunch and of the diaphragm. There is, in consequence, escape of gas and food into the abdominal cavity, which, however, only once in a way gives rise to peritonitis, as such cases terminate, as a rule, rapidly. The peripheral vessels, as well as the great veins (jugular and axillary) are gorged with blood, which is dark red, thick, sticky, coagulates badly, and assumes a bright red colour when exposed to the air. The lungs are very hyperæmic, and are interspersed with subpleural and parenchymatous hæmorrhages. Pulmonary œdema is occasionally present. We also find hæmorrhages under the costal portion of the pleuræ, especially in the upper and anterior regions. The right side of the heart is considerably filled with blood ; the coronary veins are injected, and hæmorrhages are found under the epicardium and endocardium. Rupture of the right wall of the heart occurs in rare cases. In the intestinal canal we find hyperæmia of the mucous membrane and submucous and subperitoneal hæmorrhages. Rupture of the vessels of the spleen and destruction of its tissue have been observed. The mucous membranes of the head, cerebral membranes and sinuses are very hyperæmic, and apoplectic foci are sometimes found in the brain.

Symptoms.—The most striking symptom is the sudden and rapidly developing increase in the size of the abdomen, especially in the left flank (position of the rumen), which is more or less prominent. In very severe cases this distension may be so extreme, that the left flank may rise to the level or even project above the spinal column.

The abdominal walls are tense and very elastic to the touch, and percussion produces a full, clear, tympanitic sound, which may even be metallic. There are no normal sounds of the rumen. From the beginning of the attack appetite and rumination are suspended and, despite severe straining of the animal, defecation ceases after small quantities of dung have been repeatedly passed. The animal stands with the legs either placed under the body or spread out; the back is frequently arched; the tail raised, the ears hang down in a limp condition, and the animal can be made to move only by considerable urging.

With increasing tympany, the breathing becomes more accelerated and laborious; the animal grows anxious, excited, and totters hither and thither; the visible mucous membranes of the head, and particularly the conjunctiva and the vessels of the sclerotic, are greatly injected; the superficial veins, especially of the head and throat, and the milk veins are engorged and very prominent; the eyes stare; sweat breaks out at the base of the ears, behind the elbows and on the flanks; the pulse becomes more and more frequent and wiry (small and hard), and finally imperceptible; the heart-beat more palpitating; dyspnœa increases, and respiration is short and quick, while the nostrils are distended, and even the mouth is kept open and the tongue stretched out. The animal groans, moans, slavers, froths at the mouth, sometimes coughs slightly, stretches itself out, and manages by great exertion to make a few short and interrupted eructations. In rare cases vomiting occurs. The limbs become quite cold; the mucous membranes cyanotic (of a dark-blue or leaden colour); the animal stands stupefied and motionless, gradually begins to stagger in its hind-quarters, falls, becomes convulsed and soon dies.

Pathogenesis.—The symptoms are easily explained. They are generally due to poisoning by carbonic acid, and in rare cases to cerebral apoplexy. The carbonic acid poisoning is produced partly by compression of the lungs caused by the forward displacement of the diaphragm, and partly by the absorption in the blood of carbonic acid gas from the stomach. The congestion in the anterior half of the body (injection of the mucous membranes, hyperæmia of the brain, and venous congestion), arises from pressure of the tensely filled rumen against the large main arteries of the posterior part of the

body, and from impediment of the return of the blood to the heart and lungs. Cerebral apoplexy and the formation of ecchymoses may result from excessive engorgement.

Course and Prognosis.—The course of acute tympanites is very rapid. In severe cases the animal may die in a few hours, and even in half an hour, if help be not speedily procured. A perfect cure is not always obtained by the mere removal of the accumulated gases; because the fermenting food may continue to generate fresh supplies for from twelve to twenty-four hours. If the inflation be moderate, a spontaneous recovery may sometimes take place, in consequence of abundant eructations and intestinal evacuations. Where professional assistance is not obtained at once (and, owing to the rapid course of the disease, this is not always practicable), the prognosis will be more or less unfavourable, and will depend largely upon the number of affected animals in a stable or herd.

Treatment.—The method of prophylaxis in acute tympanites may be inferred from the nature of the causes to which it is due. Great care should be exercised in changing from dry to green food. At first we should avoid luxuriant pastures, and, before taking cattle out, should give them some dry food, so that they may not begin to eat green stuff on an empty stomach; should keep them only for a short time on clover lands, and watch for any appearance of tympanites. Precautions should be taken to prevent cut green fodder from fermenting. If practicable, an œsophageal tube, trocar and canula should be kept in readiness in case of accident.

In almost all cases the removal of the gases is the first consideration. Among rough and always available methods of treatment, we may mention pulling the tongue repeatedly out of the mouth, by which means eructation is produced; and drawing a knotted straw band to and fro in the mouth, which brings on eructation, and sometimes even vomiting. A similar effect is produced by gagging the animal with a straw rope smeared with cart-grease, tar or other nauseous stuff, and tied behind its horns. For the same purpose, butter or lard (cattle ½ lb. and sheep 1 oz.) is greatly used in some parts of France.

In mild cases the paunch may be cleared of gas by movement. Driving the animal up-hill has been suggested for the

removal of wind, but such measures should be very carefully employed, as, where there is a great accumulation of gas, the condition may be rather aggravated than improved.

Rychner justly calls strong and steady pressure on the left flank the "safest," "cheapest," "most convenient," and, we might add, "most harmless" means for reducing inflation, especially in mild cases. In performing this operation, we can feel how the pressure of the hand stimulates the movements of the rumen, after the restoration of which eructation often takes place quickly. This continued pressure not only gives rise to a reflex stimulation of the gastric nerves, but the arteries in the walls of the distended paunch being emptied of blood, wherever there is pressure from without it relieves that of the accumulated gases within, arterial blood is re-conveyed to the compressed spot and a local peristalsis is restored. This movement is conveyed to the rest of the wall of the stomach and conduces, finally, to eructation and the expulsion of wind. The paunch may be completely emptied of gas in this manner. The same effects may be obtained by massaging the flanks, kneading, striking the left flank with the hand, compression of the abdomen by means of a rope wound round the trunk in spirals, bathing the left stomach-pit with cold water or cold-water bandages may all be tried. The best method is, however, to exercise a continued pressure with both hands in the neighbourhood of the paunch. The expulsion of wind may be further expedited by the mechanical removal of the contents of the rectum and the administration of irritant enemata. Many internal remedies are used for acute tympanites, but such remedies are only likely to be of service in slight cases, and in these the mechanical measures detailed above generally suffice. The water present in the draughts tends to increase the process of fermentation: there is also a danger when drenching the animal, of the mixture entering the air-passages. And should it be necessary to resort to slaughter, a large number of the proposed remedies would render the flesh unfit for consumption. The drugs employed are stimulant, emetic, absorbent, anti-fermentative and purgative in character. The best of these measures undoubtedly are: brandy (½ to 1 pint in twice the amount of cold water), tincture of veratrine (½ oz.) in combination with brandy, and veratrine (¾ gr. to 35 grs. in a spirit solution) administered subcutaneously. Tincture of colchicum (1½ drs.) is also recommended, as are the carminatives, though their action is weaker. Among the latter may be mentioned

the infusions and decoctions of cumin, fennel, anise, valerian, camomile, garlic, pepper and ginger. Oil of turpentine (2 oz. to 7 oz. in brandy and water) may be tried, although it is not reliable. Eserine should not be employed on account of the disagreeable by-effects in cattle suffering from tympanites. On the other hand, large doses of aloes (1¾ to 3 ozs.), and barium chloride (2½ drs. to 3¾ drs. in a bottle of water) are strongly recommended. The absorbents are of but little use in this condition. Ammonium chloride dilute (½ oz. to 1 oz. in 30 to 50 times the amount of cold water) is harmful ; the draught is extremely likely to find its way into the air-passages and its action is more stimulant than absorbent. The other absorbents, such as concentrated soap and water, alkaline ashes, potash and lime water are of a more harmless nature. In employing these so-called absorbents, it should not be forgotten that the conditions in the paralysed stomach differ considerably from those which are obtained in the laboratory, where the action of absorbents introduced into a test tube of carbonic acid gas may be hastened by repeated shaking, and that the carburetted hydrogen (CH_4) can under no conditions be absorbed. Anti-fermentatives should also only be exhibited in slight cases of tympanites. Of these the most useful are: sodium hyposulphite (¼ lb. to ½ lb.) ; potassium chlorate (1 oz. to 2 oz.) ; solution of chlorinated soda (2 oz. in ¾ pint of cold water) ; hydrochloric acid ; creolin ; and lysol.

On approaching danger, we should resort without delay to the employment of the œsophageal tube and to puncture of the rumen. A full description of their technique is to be found in the surgical text books. The introduction of the tube cannot always be managed without risk, especially when the animal is very restless and suffers from dyspnœa. The escape of gas is sometimes prevented by the tube getting plugged up with food, in which case nothing is left but puncture of the rumen. It should be borne in mind that a slit made with a scalpel in the outer skin will considerably facilitate the operation. A round unfenestrated trocar is the best for this purpose. In order to guard against a too rapid flow of blood to the abdominal cavity, the escape of intestinal gas should be interrupted at intervals. Such hyperæmia in the gastric region being compensated by a corresponding withdrawal of blood from the cerebral region, swooning may follow and the animal fall down. The trocar should not be taken out too soon, because the continued fermentation of the contents of the rumen may

set up renewed inflation. The canula, which is closed with a cork, should be allowed to remain in for some hours longer, during which time it should be continuously watched, so that it may not fall out. After the operation, lime water, etc., may be injected through the trocar directly into the rumen (Hansen). In default of a trocar, the operation may be performed with a knife. After the primary danger has been averted, it is imperative to keep the animal short of food for some days, because the retained fermenting food might bring on another attack.

Therapeutics of Acute Tympanites of Sheep.—The treatment is nearly the same as that for cattle. We should chiefly rely on pressure upon the left flank and on massage, while the fore-quarters of the animal are kept in a raised position. The use of the œsophageal tube cannot be recommended with sheep, and it is manifest that puncture of the rumen is more dangerous with them than with cattle. We have, therefore, to rely more on internal remedies than when treating large ruminants, and may give half-pint doses of lime water at intervals of 10 to 15 minutes, sulphate of soda (½ oz. dissolved in a little water), spirits of camphor (a dessert-spoonful) every quarter of an hour, tincture of colchicum (5 drops), oil of turpentine (half a tablespoonful in ¼ pint of oil), and paraffin oil (half a tablespoonful in the same quantity of spirits). We consider the administration of ammonia to be as hazardous with sheep as it is with cattle. If we have to deal with a large number of patients, individual medicinal treatment will generally be impossible, and we may consequently be compelled to adopt other methods, such as exercising them, or driving them into water in which they can be dipped, or the water poured over them. In desperate cases, puncture of the rumen with the trocar, or, failing it, with the knife, will often be the only remedy left.

Acute tympanites of pigs is often observed after the consumption of fermenting food, such as the grains of white beer, which are very rich in yeast; after too copious drinking of warm, sweet whey, after feeding on stale, badly-cooked carrot-tops, after poisoning, etc. As the administration of fluid medicines is accompanied by great danger, the use of the trocar will be the most reliable means of treatment. Veratrum, employed subcutaneously in doses of 1 to 3 gr., may be of benefit.

Acute tympanites of dogs occurs in some cases of poisoning (toxic gastritis and enteritis), and in intestinal stenosis.

CHRONIC TYMPANITES OF RUMINANTS.

Nature.—This chronic tympanites consists in a persistent, obstinate and continually repeated accumulation of gas in the rumen ; but not to such a great extent as in acute tympanites. Erdmann found that the composition of the gases in one case was as follows :

Carburetted hydrogen ..	..	42 per cent.
Carbonic acid ..	.. ..	35 ,, ,,
Nitrogen ..	.. .. ..	20 ,, ,,

Lungwitz obtained similar results, with traces of $S.H_2$. According to him, the gases are chiefly composed of marsh gas and carbonic acid in the proportions of 1·1 to 1·2–1·5, and consequently, there is less carbonic acid in chronic tympanites, than in the acute form.

Causes.—Among the causes of chronic tympanites there are two of equal importance, though they are dissimilar in character. Very often it is only a symptom of chronic gastric catarrh, brought on by inactivity of the rumen, cessation of rumination and consequent increase of fermentation. Should chronic gastric catarrh be present, the slightest errors in diet, such as eating too quickly or too much, and, in young animals, the taking of too much milk, etc., may easily give rise to the formation of an abnormal amount of intestinal gas. This is especially likely to occur where fodder which is apt to set up fermentation is given over long periods. The result may easily be brought about by chaff which is cut too fine, too much meal, and a consistent lack of rough fodder.

Secondly, mechanical impediments to rumination and eructation, such as stenosis of the gullet from new-growths and abscesses. Thus, chronic tympanites, brought on by tubercular degeneration of the lymph glands of the mediastinum, is not infrequently a characteristic symptom of tuberculosis. Sarcomata and actinomycomata of these glands produce a similar effect. New-growths (polypus, papilloma, fibroma, sarcoma), balls of food, balls of sand and hair-balls also prevent the escape of gas. Atony of the gastric muscular apparatus, especially that of the paunch and reticulum, adhesions of the stomach to the abdominal wall, abscesses in the walls of the stomach, prevent the former from contracting, and thus set up fermenta-

tion. These peritoneal adhesions and abscesses are generally caused by sharp-pointed foreign bodies in the stomach, blows or thrusts on the inferior region of the stomach, and repeated punctures of the paunch (see remarks on traumatic indigestion, page 108). Ruptures of the diaphragm, with prolapse of the reticulum into the thoracic cavity, and, possibly, adhesion to the thoracic organs, might greatly interfere with rumination and eructation, and might then produce chronic tympanites. This affection has also been observed as a consequence of intestinal stenoses (tumours) and of tape-worms (*tænia denticulata*).

Symptoms.—The symptoms of chronic tympanites are essentially similar to those of chronic gastric catarrh. The principal symptom is a gradually appearing, periodic, intermittent and, finally, obstinate and persistent, though moderate distension of the hollow of the left flank. At the same time rumination is suppressed, the movements and sounds of the rumen are indistinct, and occasionally vomiting is observed. Usually, there is little or no dyspnœa or restlessness. In the further course of the disease, which often lasts for months, chronic diarrhœa and emaciation may be seen. With respect to prognosis, we find that tympanites produced by chronic catarrh of the stomach disappears as soon as its cause is removed, but tympanites due to organic changes and displacement of the stomach is incurable. Tuberculosis of the mediastinal glands may be demonstrated by tuberculin.

Therapeutics.—Treatment is successful only with the first mentioned form and, in this case, is similar to that of chronic gastric catarrh. We should restrict the fermentation by antiseptics (hydrochloric acid, hyposulphite of soda, creolin and neutral salts), and should stimulate the peristaltic action of the stomach by agents such as tartar emetic, white hellebore, oil of turpentine, aloes, barium chloride, etc. Trasbôt has obtained good results from ipecacuanha in doses of 1 to 2 drachms. Eserine cannot be recommended, because of its unfavourable secondary effects. As a last resource in severe cases, we may make an incision in the left flank, and remove the accumulated fermenting food, and may also employ a permanent canula. The use of the trocar for removing the tympanitic gases is rarely required in the chronic form of the disease. The gas-absorbing remedies have only a temporary influence, and

are therefore useless. Regulation of the diet is, however, of the greatest importance.

IMPACTION OF THE RUMEN IN CATTLE.

Cause.—The condition known as impaction of the rumen (over-eating or over-feeding), which is frequent among cattle, is generally caused by the too rapid consumption of an unusually large quantity of food that is appreciated by the animal, such as green fodder, new hay, fresh clover, different kinds of brewers' grains and mashes. It also arises from natural greediness, fasting for too long a period and from drinking too much water, especially when the fluid is cold.

Symptoms.—These vary according to the quantity, quality, and digestibility of the food that has been eaten. They are usually as follows:—The animal stands away from the feeding trough, arches its back, passes at short intervals and in small quantities normal dung, whisks its tail, kicks with its hind legs against the abdomen, looks round at its hind quarters, while moaning and groaning, occasionally shakes its head violently, and expels air through the nostrils with a hissing sound. Or it stands stupidly and listlessly, without taking any heed of its surroundings. On rare occasions it may lie down and groan, but get up again after a short time. Appetite is lost, rumination is entirely suppressed, and thirst is sometimes increased. Salivation is often present, in which case the animal goes through the motions of eating without any food in its mouth; occasionally short interrupted eructations escape while the head and neck are stretched out; or peculiar retching movements and true vomiting may take place and be frequently repeated. The muzzle is generally normal. The abdomen, especially on the left side, is greatly increased in circumference. The hollow of the left flank is obliterated and may even project upwards. On palpation of the rumen through the wall of the flank, we find that it is tense, filled up sometimes with doughy, yielding, at other times, very firm chyme, which usually reaches to the upper wall of the rumen, so that percussion gives an empty sound all over this part. Later on, a slight accumulation of gas above the food-contents of the rumen may be found by palpation and percussion. If strong and continued pressure be made on the rumen, the animal will show that it is disagree-

able, and try to avoid it. The condition of the paunch can be ascertained by exploration through the rectum, instead of by palpation of the flank. The movements of the rumen, which can be seen and felt, when that organ is in a normal condition, are entirely suppressed, or so much diminished that the hand, when laid on the paunch, can feel no activity in it, and its sounds are greatly diminished or inaudible. There is more or less constipation, and the dung, instead of being passed in flat cakes, is firm, balled and of a dark colour.

The temperature is not increased, and is uniformly distributed; but the pulse is somewhat accelerated and hard. Respiration, on account of the pressure on the diaphragm, is laborious, and when the animal is recumbent or going down hill, becomes so audibly distressed, that an affection of the lungs might be suspected. The expression is anxious and staring, the eyeballs are prominent, and the conjunctiva is injected.

Course.—The intensity of the disease varies. If the rumen be greatly overloaded, symptoms of suffocation and apoplexy may quickly appear, as frequently happens in acute tympanites. In other cases an acute gastro-intestinal catarrh becomes developed. The attack usually passes off, especially if the animal was previously healthy and strong, the quantity eaten not too great, nor the food too indigestible. In mild cases recovery may take place in a few hours, and signs of digestion appear within the first twenty-four to thirty-six hours. Rumination and appetite are regained, the movements of the paunch become active, frequent eructation of gases and copious defecation take place, and in from three to four days recovery is complete.

Differential Diagnosis.—Overfilling of the rumen may be confused with acute tympanites and, less frequently, with pelvic hernia. The mistake arises, in the former case, because acute tympanites appears very rapidly, and directly after feeding, and shows increase in the size of the paunch; and in the latter, because the hernia occurs suddenly, and gives rise to symptoms of uneasiness. An exploration of the rectum guards us from a confusion with ventral hernia; because in impaction of the rumen there is no tense cord in front of the entrance to the pelvis, on its right side towards the abdominal ring. The history of the case is also a good guide. In

tympanites the distension of the rumen is greater, and we find by palpation an accumulation of gases and not of firm food. The gas developed in cases of impaction of the stomach, exists only in small quantities and, as a rule, soon passes off; but the disturbances of respiration are of a more severe character. Torsion of the uterus may also simulate impaction of the stomach; because it causes sudden refusal of food, inflation of the left flank, and constipation. Beel and Johne advise that an examination *per vaginam* should be made in pregnant cows which show symptoms of impaction. An accidental confusion with acute gastro-intestinal catarrh can be easily avoided by paying attention to the history of the case (sudden appearance of severe symptoms). Even the layman seldom fails to recognise impaction of the rumen.

Treatment.—In slight cases a purely expectant method of treatment will suffice, and nothing further need be done than to keep the cattle short of food for several days, and to exercise them well. In severer cases we should try to stimulate the rumen externally by massage (pressing, kneading and rubbing from the left flank), and internally by aperients and emetics. Aloes, tartar emetic and sulphate of soda are given in large doses. Here, the best emetic is white hellebore root, (150 to 300 grains; or tincture, ½ oz). Trasbôt states that ipecacuanha is of very great value, either at the beginning of the attack, or to complete the effect of puncture. As a last resource, we may make an incision into the rumen and remove a portion of the accumulated food.

VACUITY OF THE RUMEN.

Under the name of "Vacuity of the Rumen," or "Railway Sickness," Voigtländer has described a peculiar and very obscure morbid condition of cows far advanced in pregnancy, which was produced by rapid and almost entire removal of the contents of the paunch. The animals in question had been carried for a long distance by rail, during which time no food was given, and previous to departure they had only a small quantity of grass, which was digested at an early period of the journey. According to Voigtländer, the symptoms closely resembled those of paralytic milk fever. The cows, being completely paralysed, were unable to stand; the head was turned to one side; the eyes were deep-sunken and dim; the pupils dilated and respiration greatly accelerated. Only a few recovered. On *post-mortem* examination, the rumen

was found to be so empty, that it could be wrapped round the arm like an empty bag. Viegel found the same symptoms in cows that had suffered from similar conditions. The cows, especially those that were in good condition, became ill soon after their arrival, and died in from eight to twenty-four hours. The most prominent symptoms were : High fever, complete loss of appetite, continued lying down and a paralytic condition of the organs of digestion. *Post-mortem* examination showed that the rumen was inflated and nearly empty; the liver hyperæmic or clay-coloured, the small intestines very red; and peritonitis and hyperæmia of the uterus were sometimes present. Weigel ascribes the disease to long-continued empty condition of the paunch (for two or three days), want of rest from not being allowed to lie down, and spinal concussion. Röder, Konig, Noack and Schmidt report similar cases. They also consider that the results are due to long journeys in a standing position, in hot railway carriages and without either food or drink. Other characteristic symptoms are said to be disturbances of equilibrium and paralysis of the bladder. *Post-mortem* examination reveals no very decided changes. The musculature is said to become a dirty brown-red to brown-black and to show fatty degeneration (Noack). Investigation has up to the present offered no certain information as to the nature of this condition, to which the name of "vacuity of the rumen" has been given. It is probable that it is a parenchymatous myositis resulting from over-fatigue and analagous to the over-strain and degenerative muscular atrophy frequently seen in horses after parturition. Villagio has recently expressed similar views ; he explains this so-called "railway sickness" as due to muscular over-strain, the primary affection being muscular, and the secondary nervous.

VOMITING.

Vomiting occurs in impaction of the rumen, in some affections of the œsophagus (*diverticula*), in invagination (intussusception) of the intestine, and from the use of eserine, eseridine, pilocarpine, arecoline, veratrine and apomorphine. In other cases the causes of vomiting are unknown.

According to Strebel, chronic vomiting is, with few exceptions, a very dangerous symptom in cattle. He considers that it is provoked only by absolutely incurable pathological conditions of the glands of the mucous membrane of the stomach, œsophagus, or diaphragm. Such are : impaction of the rumen ; abnormal sensibility and inflammatory conditions of the mucous membrane of the stomach ; ulcers ; constriction of the pylorus ;

prolapse of the reticulum due to rupture of the diaphragm; swelling of the mediastinal glands, etc.

CATARRH OF THE ABOMASUM AND SMALL INTESTINE IN CATTLE.

As already stated, the four compartments of the stomach of ruminants may be respectively attacked by a primary catarrhal inflammation. Thus it seems that the abomasum is not uncommonly the seat of a primary affection, the correct diagnosis of which is generally very difficult. Prietsch found among 600 apparently healthy cattle which were slaughtered, from 20 to 25 per cent. that had either acute or chronic catarrh of the abomasum. In the majority of slaughtered cattle Ostertag found under the epithelium of the mucous membrane of the abomasum a parasite, the *strongylus convolutus*, which he regards as the cause of emaciation and hydræmic cachexia, when it invades that organ in large numbers. As descriptions of catarrh of the abomasum are contradictory in some essential points, we shall content ourselves with briefly referring to observations which are least in dispute.

Saake has met with catarrh of the abomasum in young cattle, oxen and cows before and after calving. In the beginning the symptoms are the same as in other gastric affections. During a series of alternate improvements and relapses gas becomes frequently developed in the rumen, although the animals may look remarkably well, in spite of considerable tympanites. On auscultation of the rumen we may hear a peculiar and frequently loud metallic sound, which Saake considers to be a characteristic symptom of catarrh of the abomasum, as it is never heard in a case of ordinary inflation. Later on, severe diarrhœa sets in, with gradual emaciation followed by a chlorotic condition, and a tendency to œdema in the intermaxillary space. The appetite becomes vitiated and capricious, without well-marked pica being present; and the right hypochondrium becomes sensitive to pressure. The course of the disease is very long, and often ends in death after several months. On *post-mortem* examination, we find considerable swelling of the mucosa and submucosa, which are œdematously infiltrated and sometimes 1½ inches thick at the pylorus and beginning of the duodenum; and catarrhal reddening, especially in the pylorus, and in chronic cases follicular ulcers of the mucous membrane. Tannin in doses up to 1½ oz. has proved very effective in this disease. Under the name of "Catarrh of the Abomasum and Small

Intestine," Harms has described a suddenly appearing affection of the organs of digestion, which takes either an acute or a chronic course, and is distinguished from ordinary gastro-intestinal catarrh by icteric colouring of the conjuctiva, normal condition of the fæces, unimpaired activity of the rumen (even when it is filled), and manifestations of pain, on pressure being applied to the region of the abomasum. Later on symptoms of pica make their appearance. On *post-mortem* examination of the early stages, we find œdematous swelling, hyperæmia and a mucous coating; and in those of older standing the mucous membrane is thickened and has abnormally large folds. As regards treatment, Harms recommends low diet, easily digestible food and the administration of Carlsbad salt and hydrochloric acid. He has described a case of impaction of the abomasum, which contained 78 pints of ruminated food and, consequently, if viewed from a distance, might have been mistaken for the rumen in an inflated condition. The symptoms during life were frequent eructations, pain on pressure being applied to the region of the abomasum, salivation, vomiting and great obstruction of the rectum by fæces. According to Dickerhoff, impaction of the abomasum seldom occurs in cattle. It is generally caused by over feeding, especially with tough, mouldy or spoilt food. It is dangerous only when severe, and it is best treated by purgatives in large doses. Cuille and Sendrail found a sarcoma in the abomasum of an animal which had suffered from chronic digestive disturbances and had died under pronounced symptoms of marasmus. There was thickening of the gastric wall in the neighbourhood of the pylorus to the extent of four inches. Kitt has described a case of scirrhus of the abomasum.

GASTRO-INTESTINAL CATARRH OF YOUNG CATTLE.

Gastro-intestinal catarrh of young cattle (sucklings and newly-weaned calves) deserves to be discussed separately, as it is a subject of great importance to breeders of cattle and graziers, and is essentially different from the same disease in grown-up cattle, both as regards its causes, course and treatment. Added to which it is frequently confounded with infectious or "White Scour," and will be referred to again under that heading. It is expedient that simple gastro-intestinal catarrh in young cattle, as opposed to infectious dysentery, should be dealt with in a separate chapter.

Etiology.—(*Causes of the Disease in Sucklings*).—1. Diseases and abnormal conditions in mothers. The physical state of the cow is closely connected with the health of the suckling

by means of the milk, particularly with regard to digestive diseases, mal-nutrition, cachexia and debility. Infective diseases, such as foot-and-mouth, general tuberculosis, and tuberculosis of the udder, are most dangerous. Mastitis and even temporary inflammatory swelling of the udder very often cause gastro-intestinal catarrh in the calf. The feeding of the cow is also of the greatest importance. Food which is too fattening, too concentrated and too watery, or food which is decomposing and otherwise unwholesome, may produce indigestion in the calf, in consequence of their bad effect on the milk. In this respect the chief lacteal faults are—too much or too little fat in the milk; too little or too much casein; tendency of the milk to coagulate in the udder; and a bitter, rancid or ropy condition of the milk. Foreign substances (such as ethereal oils, resinous and pungent matters, poisons and medicines, especially aperient salts) in the milk may exercise an irritating effect on the mucous membrane of the calf's stomach. Over-exertion on the part of the cow may also affect the health of her young one, by impairing the quality, or diminishing the quantity of the milk. Some authorities consider that the usual practice of removing the colostrum by milking is a cause of this disease.

2. Errors of diet during lactation. Here the most frequent cause is the consumption of too large a quantity of milk at one sucking, after the calf has been separated for a comparatively long time from the cow, and has consequently become unusually hungry. The giving of milk-substitutes may also bring on catarrh.

3. Chills received by the newly-born calf, from a cold and damp stall, and from a cold udder.

4. Retention of the meconium, in which case the resulting constipation leads to decomposition of the intestinal contents and irritation of its mucous membrane by the products of decomposition.

Causes of the Disease in Newly-Weaned Calves.—

1. Faulty weaning, such as too early feeding on dry food, especially coarse, fibrous and irritating fodder, which can hardly fail to produce a morbid condition of the stomach and intestines; mouldy forage and straw which has been cut too short.

2. Improper milk-substitutes, particularly those which are too rich in starch. They set up inflammation of the intestinal

mucous membrane by the production of acetous, lactic and butyric fermentations.

3. Bad weather, especially during the spring and autumn; and drinking too cold water.

4. Shedding of the milk-teeth, during which time the calves gnaw and swallow all kinds of objects.

5. The presence in the alimentary canal of worms (as for instance, strongylus contortus in stomach-worm plague of lambs; tænia expansa in tapeworm diseases of lambs; and strongylus armatus in sucking foals). Some animals are predisposed to gastro-intestinal catarrh from congenital weakness of digestion, diseases of the mesenteric glands, etc. Every newly-born animal is liable to it, in consequence of the great sensitiveness of its organs of digestion.

Anatomical Conditions.—On the whole, the anatomical changes in the stomach and intestine are the same as those met with in adults, except that in fatal cases they are not always in proportion to the excessively severe symptoms exhibited during life. The contents of the stomach of sucklings usually consist of firm, lumpy, caseous clots; and those of newly-weaned animals, of balls of coarse hay and straw. In sucklings the normal coagulated condition of the stomach is not uncommonly regarded as abnormal. The gastro-intestinal mucous membrane shows various catarrhal changes, such as congestion, swelling, follicular catarrh, and even ulcers in cases of acute catarrh; and atrophy, thickening, prominences, pigmentation, etc., in the chronic form. The mesenteric glands are generally more or less swollen. In many cases the carcases are greatly emaciated and show signs of anæmia and hydræmia.

Symptoms.—The symptoms may appear either gradually or suddenly, according to the cause. In sucklings, diarrhœa, which is the principal symptom, may be preceded only by decreased inclination to suck, slight debility and depression. If the disease be due to errors of diet or to chill, the symptoms, especially in newly-weaned animals, will appear suddenly in the form of fever, well-marked constitutional disturbance, unequally distributed temperature, cold legs, dry muzzle, debility and depression. The diarrhœa, which soon sets in, consists at first of thick pasty fæces of normal colour and smell; later on, it becomes watery and light coloured, and is frequently expelled in jets; and, finally, it is greasy, yellow or dull-green, frothy,

sour-smelling, or very fœtid and intermixed with flakes and lumps. Towards the end the dung shows streaks of blood, or may assume a sanguineous colour; is passed involuntarily; soils the hind legs and tail; and by its corrosive action, frequently causes the hair in the neighbourhood of the anus to fall out. Tenesmus and symptoms of slight colic are present; the animal stands with its back arched and the hind legs placed under the body; the skin is dry; the hair stands on end; the effluvium from the body is very fœtid, and the animal becomes excessively faint and weak. In combination with the diarrhœa we often observe the repeated appearance of tympany of the paunch and intestines. This inflation is generated by abnormal decompositions which occur during the course of the gastro-intestinal catarrh, and may become developed before, after, or simultaneously with the diarrhœa.

The course of the disease may be either acute or chronic. Death may occur in a few days, or may be deferred for several weeks. In most cases death is due to exhaustion, general anæmia and hydræmia. The disease is sometimes complicated with catarrhal pneumonia, which is probably a result of atelectasis (imperfect expansion), as may frequently occur in weak animals immediately after birth. Simple intestinal catarrh can be distinguished from dysentery by its more sporadic occurrence, its milder character, its more favourable course, and the fact that it does not appear so soon after birth as infective dysentery.

Prognosis.—In this disease prognosis should always be made with caution, especially if the diarrhœa and tympanites are of long duration. Otherwise, the prognosis depends upon the nature of the originating cause.

Treatment.—In this respect diet occupies the first and medicine the second position. With the object of removing the cause in a suckling, treatment should be chiefly applied to the mother. All factors which unfavourably influence the quality of the cow's milk, especially too fattening food, bad maintenance, and too long retention of the milk in the udder, should be removed. If the illness of the calf can with certainty be traced to the cow, the former animal should be withdrawn from the pernicious influence of the latter, by putting it to another cow which is healthy. If the diarrhœa is due to improper feeding, the error should be corrected without delay. With sucklings care must be taken that they do not consume too much milk at a time, little and often being the best rule

with them, because their stomachs are not suited for the digestion of large quantities. If we cannot allow the calves to suck when they like, we should give them an opportunity of doing so at least three or four times daily. With calves about to be weaned, we must proceed gradually. For instance, calves should not be weaned before the age of from four to six weeks, and lambs from three to four months. In both cases the process should be carried out gradually, and all stimulating and indigestible food avoided.

To combat the diarrhœa we may at first use slightly astringent and mucilaginous domestic medicines, such as toast, parched meal, coffee, raw eggs, lime-water; mucilage of althea, or gruel; milk of almonds, of hemp-seed and of poppy-seed; infusions of hops or of poppy-heads. We may begin with a slight aperient, especially if fermenting substances have accumulated in the intestines. To calves we may give two or three tablespoonfuls of castor oil or linseed oil; or 1 oz. of Glauber's or Epsom salts, in a mucilaginous infusion (quarter of the dose for lambs). Soluble cream of tartar has been recommended as a laxative (calves ¾ to 1 oz., and lambs 75 to 150 grains).

For abnormal fermentations in the intestinal canal, we may give creolin (calves 1 dr., and lambs 10 to 15 min.) in cold water two or three times a day. Also, carbonate of magnesia, carbonate of soda, chalk, bone meal, creosote (calves 3 to 5 drops, lambs ½ to 1 drop), tar (in the form of tar-water by the mouth, or as an enema), salicylic acid (calves 15 to 30 grains, lambs 4 to 8 grains in spirit), resorcin, lysol, the ethereal oils, and other antiseptics have been found efficacious.

In severe cases of diarrhœa, opium (powder, tincture, or as Dover's powder) and rhubarb render good service. Opium may be given (15½ to 46 grs. for calves, 1½ to 3 grs. for lambs), or as tinctura opii simplex or as Dover's powder (1¼ drs. to 2½ drs. for calves, 15½ grs. to 30 grs. for lambs). Rhubarb-root is best given as an infusion (30 grs. to 75 grs. for calves, 7¾ grs. to 15½ grs. for lambs). The other astringents and styptics may also be employed. Tannin, tannoform, tannalbin, tannopin (46 grs. to 75 grs. in repeated doses), have been recently recommended in this condition. Tannopin (Schindelka), and tannoform (Bass and others), when given in repeated doses of 30 to 45 grs. are said to be excellent in the diarrhœa of older sucklings and newly-weaned calves. The irrigation of the intestines with astringent solutions is also recommended. We may try enemas of a one per cent. solution of alum or tannin.

TRAUMATIC INDIGESTION IN CATTLE.

General Remarks.—The literature on the subject of disturbances caused by foreign bodies in the stomach of ruminants is very rich. In most cases it refers to so-called traumatic carditis and traumatic pericarditis, which are complications or stages of the purely gastric affection that we are at present discussing. Traumatic disease of the heart will be dealt with later on (p. 409). Although the cardiac condition presents a more significant and characteristic clinical picture and has, up to now, formed the bulk of the material included under the heading "Foreign bodies in the stomach in ruminants," it is also possible to give a broad general impression of the gastric and diaphragmatic disturbances which the presence of foreign bodies is likely to engender. Foreign bodies are found very frequently in the stomach of ruminants, especially in cows, and without any previous manifestation of disease. A carving knife is said to have remained three months in the stomach of a beast without causing any injury. In other cases, foreign bodies produce more or less grave lesions in the reticulum and adjoining organs, particularly in the diaphragm. The intensity of the affection depends, above all things, on the external condition of these bodies. The following is an appropriate classification :—1. Blunt bodies, as, for instance, stones. 2. Pointed bodies, which, like nails that have a head, penetrate the wall of the stomach and remain fixed in it. 3. Pointed bodies which pass completely through the wall of the stomach, as, for example, needles and pieces of wire.

Etiology.—There are several reasons why foreign bodies are found more frequently in the stomachs of ruminants, and especially in those of cattle, than in other domestic animals. Cattle have the habit of licking objects within reach and swallowing them if possible. They consume food very rapidly and chew it imperfectly, leaving its comminution to be completed during rumination. Cows with female attendants, with their hairpins and needles, are more liable to suffer from traumatic inflammation of the stomach than cows which are tended by men. The disease is met with more rarely on large farms than on small holdings where kitchen refuse of every kind is used for cattle-food. The inner wall of the stomach of cows kept by shoemakers is sometimes almost paved with nails. The disease is also comparatively common in the neighbourhood of stocking and needle factories and in byres with shingle roofs. It is more frequent in stalls than on pasture. Harms found that animals

which stand at the lower end of the feeding trough are more apt to swallow foreign bodies, which readily accumulate at the sloped end.

Anatomical Conditions.—The *blunt* foreign bodies which are found in the reticulum are usually stones or sand ; but we may also find balls of hair, articles of clothing, pieces of leather, straps, broken handles of whips, balls, spoons, coins and even nakes. These bodies produce catarrhal or inflammatory processes in the mucous membrane of the stomach. In impaction of the stomach with sand, the grains of silica form a hard, compact mass, and the mucous membrane is densely incrusted with sand.

The pointed foreign bodies which do not pass completely through the wall of the stomach are usually nails with a head, pins, hair-pins and bent pieces of wire. They are generally found in the reticulum, because the honeycombed structure of its surface is peculiarly well adapted to arrest the progress of these bodies. At the place penetrated by the foreign matter, the wall of the reticulum shows a circumscribed inflammation which conceals a narrow, fistulous canal that has an indurated wall of a grey or dull-blue colour. This canal contains pus mixed with particles of food. Around this lesion the peritoneal surface of the reticulum is often adherent to adjacent organs (particularly to the diaphragm, which is also inflamed), by means of a plastic exudation, often half-an inch thick. Abscesses may be found near the adhesion ; and, sometimes, the diaphragm may show deposits of granulation tissue, which may be as much as an inch in thickness. The rumen is often adherent to the abdominal wall, and is generally filled with food. Perforating foreign bodies are very seldom met with in the omasum and abomasum. The foreign matter often quits the wound in the reticulum and re-enters that compartment. In many cases it cannot be found, on account of its having been evacuated along with the dung, a fact which formerly gave rise to the erroneous supposition that the foreign body had been dissolved.

Pointed bodies which pass completely through the wall of the stomach are generally needles of all kinds, pieces of wire, broken nails, pointed pieces of iron, knives, scissors, forks, broken bits of glass, splinters of wood, broken particles of stone, broken sticks, umbrella ribs, etc. Besides the above-described lesions of the reticulum and diaphragm, we may observe the following changes, according to the direction the foreign body has taken : extensive peritonitis, abscesses of the liver, spleen

and lungs, adhesions of the reticulum to the liver, abscesses in the thoracic and abdominal walls, lobular pneumonia, adhesions of the lungs to the diaphragm, pleuritis, etc. Sometimes the foreign body may penetrate into the spinal cord, ribs, muscles of the thigh, etc. Its passage is marked by abscesses, inflammation, formation of fistulæ and adhesions. In rare cases death by hæmorrhage into the reticulum and rumen has been observed. The changes which occur in the pericardium and substance of the heart are described under the heading of "Traumatic Carditis."

It is rare in cattle for foreign bodies to be present in the intestines. But there is an instance of a heifer which, after being unsuccessfully treated for constipation, was found to have a kitten wedged in the small intestine (Fetting).

Symptoms.—The symptoms caused by the presence of *blunt* bodies in the antestomachs of cattle are, on the whole, chronic or acute disturbances of digestion, which are due to irritation, contusion and superficial injury of the mucous membrane. Graver symptoms are found after devouring considerable quantities of sand, such as (according to Krichels-Schmidt) continuous chewing without swallowing, slavering, groaning, constipation, frequent lying down, arching of the back and gradually increasing weakness. We find on palpation that the rumen is as hard as a stone, and that the peristalsis of the stomach is diminished and finally ceases. In fatal cases, death occurs in from nine to seventeen days. Chronic indigestion is a sequel in some cases.

When the foreign body is pointed, the symptoms are generally those of inflammation of the stomach and diaphragm, and are usually as follows:—Without any apparent cause we find disturbances of digestion with suddenly developed symptoms which resemble those of invagination of the intestines, and which consist in attacks of colic, tottering hither and thither, kicking at the abdomen, etc. In other cases, only the symptoms of acute catarrh of the stomach appear at the beginning of the seizure; but subsequently a number of chronic, periodically recurring gastric attacks of greatly varying character, among which chronic tympanites is the most prominent. The fact that this symptom does not yield to any ordinary treatment is of the greatest diagnostic value. If the affection lasts for a long time, it gradually brings on great emaciation and debility. The following factors are of great diagnostic weight: The animals manifest

intense pain on various occasions by groaning, as, for instance, after feeding, when lying down, getting up, walking about, and particularly during movement, which is careful and stiff, and the expression of the face shows anxiety and pain. Usually palpation of the reticulum is very painful. The place for this palpation is on the left side of the ensiform cartilage, on the inferior abdominal wall. Frequently the animal also evinces pain on pressure of the sternum, thorax, withers and paunch. On percussion being applied to the diaphragm at its points of insertion, pain is manifested; and we sometimes find a circumscribed area of dulness in front of the diaphragm (abscesses, granulating new growths, etc.). The contractions of the diaphragm during inspiration and defecation and its movements during expiration are painful and accompanied by groaning. Hence, abdominal pressure for purposes of defecation is used as little as possible, and consequently an accumulation of fæces forms in the rectum. If a large vessel be injured by a foreign body, we may have a discharge of blood from the rectum.

If the pointed body pass completely through the stomach and diaphragm, we have other symptoms caused by its migratory tendency, from the contractions of the reticulum and the movements of the diaphragm, especially during expiration, by which the pointed body is impelled towards the thoracic cavity. In other instances it reaches the abdominal cavity by means of the peristalsis of the stomach. Strained abdominal pressure during calving, and highly-wrought action of the diaphragm in forced breathing, as in transport to and from market, usually accelerate the forward propulsion of the foreign body. According as various organs are injured by its presence, the symptoms of traumatic inflammation of the stomach and diaphragm become complicated with those of carditis, pneumonia, pleuritis, pneumothorax, enteritis, peritonitis, hepatitis, pyæmia, septicæmia and fatal internal hæmorrhage. With respect to these complications and the further course of the disease, see the remarks on traumatic pericarditis. If the foreign body finally works its way to the surface, we shall find a swelling on the affected part of the skin which will be very characteristic of this stage of the affection. This swelling is generally found on the left wall of the thorax, in the region of the left inferior ribs (intra-costal region), region of the sternum, or behind the elbow. It may appear on the right side of the thorax, and even on the abdomen, navel, flanks or udder. This phlegmonous swelling varies in size up to that of a man's head; is, as a rule, only

moderately painful; of varying consistence; and eventually forms an abscess which contains ichorous pus mixed with particles of food, sand, and occasionally the foreign body itself. These substances may be discharged by means of a fistula, through which we may sometimes pass a probe into the stomach. This fistula often does not close for several weeks, or even months, and recovery may not take place until after a long period of ill-health. In many cases, neither before nor after the suppurative process, do the animals show symptoms of general ill-health. The obstructive agent in rare cases finds an exit from the abdominal cavity by way of the uterus or in the direction of the aorta (aneurism).

There are also cases where the foreign body is retained in the contents of the first stomach, and either remains harmlessly there, or is passed out with the excretions.

Differential Diagnosis.—Diagnosis, which is of varying difficulty, can generally be made only by a careful examination, and only when the more important symptoms are present.

Confusions chiefly occur with chronic simple catarrh of the stomach, in which the manifestation of pain on palpation of the affected organs is absent; the gastric disturbances are not of such a varying nature; apparent causes are generally to be found; proper treatment leads to recovery, or at least to improvement; and emaciation is not so rapid and intense.

The distinction between traumatic inflammation of the stomach and diaphragm from peritonitis and gastro-enteritis may be difficult, although the two last-mentioned diseases are usually marked by a more rapid course, higher fever and graver disturbance of the general health. If the foreign body has penetrated into the lungs, it may produce symptoms of a chronic pulmonary affection which cannot always be distinguished from tuberculosis of the lungs, unless a previous indigestion, a demonstrable affection of the diaphragm, or the absence of the extensive bronchial catarrh which usually accompanies tuberculosis of the lungs, indicate the presence of a foreign body. The disease we are considering has also been confounded with pleuro-pneumonia, which is epizoötic in its occurrence, typical in its course, and with symptoms essentially pneumonic. It also presents no disturbances of the stomach and diaphragm. The ailment may be mistaken for anthrax, as in cases of sudden death, due to penetration of the foreign body into the heart,

to internal hæmorrhage, to peritonitis, or discharge of blood from the rectum.

Treatment.—With regard to prophylaxis, we should replace female attendants by male ones, or else properly instruct them, and frequently examine the lower strata of the food. Harms proposes to fix at each end of the feeding trough a receptacle for foreign bodies. It is most important to make an early diagnosis. So long as the diagnosis cannot be made with certainty, the treatment should be the same as that for acute or chronic catarrh of the stomach. If we know that the cause of the disease is a foreign body, we must decide between early slaughter or removal by an incision into the rumen; the former means being preferable in all severe cases. Obich's operation should be tried, if the disease be not too far advanced, although some authorities state that they have obtained good results even after pericarditis had set in. Obich's method consists in opening the rumen at the hollow of the left flank, about four inches below the transverse processes of the lumbar vertebræ, by means of a vertical incision, through which the hand can be passed into the rumen forwards and downwards, in searching for the opening of the reticulum, which is on the right hand side, and in feeling on the floor of the reticulum for foreign bodies. After removing these the wounds of the rumen and abdominal wall should be closed; the former by an intestinal suture, the latter by an ordinary suture. Usually the operation is borne very well, and may even be performed during pregnancy. It is not, however, always successful, because severe affection of the heart and diaphragm may be present, and an exact diagnosis is frequently impossible. Out of thirteen operations, Obich had only four good results. It might be tried in cases in which the diagnosis was certain and the disease not too far advanced, unless, for economic reasons, slaughter was to be preferred. Instead of this operation, Schöberl recommends that the beast be placed on its back, and the region on the left side and behind the ensiform cartilage be energetically kneaded with the foot. He states that out of sixty beasts which were treated in this way, the foreign body failed only on two occasions to return into the reticulum. Eston has obtained good results with this method in twenty-one cases. Imminger recommends the raising of the fore-quarters, in order that the diaphragm may be relieved from the pressure of the viscera and the foreign body may be thus given a chance to return.

He says that the condition may be diagnosed by the raising of the hind-quarters ; the animal lows as if in pain, while the gaze becomes distressed and painstricken.

GASTRO-INTESTINAL CATARRH OF CARNIVORA.

Etiology.—Impaction of the stomach, catarrh of the stomach, and catarrh of the intestines are far more prevalent in dogs than in horses and cattle. In the dog these maladies form about 60 per cent. of all the diseases of digestion. Among 70,000 sick dogs treated at the Berlin Canine Hospital during nine years, there were 4,000 cases (6 per cent.) of gastro-intestinal catarrh. This frequency is probably due to the careless manner in which dogs are usually fed. The common and irrational practice of giving them only one meal a day leads to a quotidian overcharging of the stomach. Hounds and bitches in milk suffer from this cause more than other dogs, on account of their greater voracity. Hungry dogs often get impaction of the stomach from eating large quantities of potatoes, bread, bones, etc. Further causes are the consumption of indigestible food (like some kinds of dog biscuits), mechanically irritating, spoiled and decomposing food ; swallowing foreign bodies (see remarks on foreign bodies in the intestinal canal, p. 121), especially by young, playful dogs, and those which fetch and carry ; imperfect teeth ; too rapid eating ; the consumption of overheated food, and chill, as, for instance, when they are clipped or sent into the water.

Acute gastro-intestinal catarrh in carnivora is an accessory symptom, especially in distemper ; as also is the chronic form of this catarrh in several chronic morbid conditions, such as ulcers in the stomach, intestinal worms, and stasis of the portal vein after chronic affections of the heart, lungs and liver and Bright's disease.

Symptoms.—1. *Overloading of the stomach* (surfeit) generally produces vomiting, which soon promotes recovery. If vomiting does not come on, the animal will retch, slaver and turn away with nausea from any food placed before him. Thirst, as a rule, will be increased ; pressure on the overfilled stomach will cause the animal to groan with pain ; respiration is often greatly accelerated. The patient is very dull, depressed and peevish, or uneasy, will frequently change its resting-place, groan, whine and show signs of colic. There is no fever.

2. *Gastric catarrh.* If a distinction between catarrh of the stomach and catarrh of the intestines be possible in our domestic animals, it can best be made in carnivora, because in the former the most important symptom, vomiting, is seldom absent. We must, however, bear in mind that it is not a characteristic symptom of gastric catarrh, because it also occurs in various other morbid conditions. Such are: gastro-enteritis; cancer of the stomach; gastric ulcer; dilatations; stenoses; incarcerations; foreign bodies in the intestines; helminthiasis; peritonitis; inflammation of the diaphragm; different throat affections, pharyngitis, and especially coughs; uræmia; septicæmia; eclampsia; epilepsy; cerebral inflammation and apoplexy; otorrhœa and poisonings.

The course of gastric catarrh in carnivora may be acute or chronic, and with or without fever. In mild cases there is nothing morbid to be seen, except dislike to exercise, depression of spirits, decreased or capricious appetite and increased thirst, which may all pass away in a day or two. Usually retching and vomiting are present. At first, the vomited matter consists of normal chyme; but later on, of ropy, glairy mucus, which contains air-bubbles, and sometimes blood-coloured or yellow streaks; and finally of more or less pure bile, especially in cases of severe vomiting. As a rule, there is great thirst, and the consequent increased consumption of water gives rise to renewed vomiting. There is little or no appetite, and any food which is taken is generally returned. There is more or less constipation. The temperature of the body is unequally distributed, the nose warm, often dry, the rate of the pulse accelerated (by 20 or more beats), and the temperature raised (sometimes to 103° F.). The animal is depressed, lies down a good deal, avoids his usual place of rest, scratches away the litter and prefers the cold floor. Pressure on the stomach is often painful.

3. *Intestinal catarrh.* If the intestine be the chief seat of the disease, vomiting will generally be absent; the appetite will be more or less impaired, and the stomach will not be abnormally sensitive to pressure. The most noteworthy symptom is diarrhœa, which is always present, owing to the shortness of the intestinal canal of carnivora, and which varies a good deal in severity. In mild cases the fæces have a pappy consistence, but in severe attacks they are watery or mucous (slimy), sometimes yeast-like, bilious, tinged with blood, occasionally very frothy, and usually fœtid; they often contain particles of undigested food, and soil the parts about the anus. Tenesmus

makes its appearance with frequent protrusion of the congested and swollen mucous membrane of the rectum. Prolapse of the rectum may occur in young and weak animals. Biliary pigments are constantly found in the urine. Fever is seldom absent in intestinal catarrh, and the temperature may rise to 106° F. Symptoms of jaundice, owing to the severity of the disease in the duodenum, is more often seen in dogs and cats than in horses and cattle.

Prognosis.—Acute gastro-intestinal catarrh in carnivora generally ends in recovery. If diarrhœa be extremely severe and protracted, very young or feeble animals rapidly lose all strength, and death may occur. Icteric symptoms render prognosis unfavourable. The fact that biliary pigments are frequently found in the urine shows that chronic catarrh probably arising from errors of feeding, is more common in these animals than is usually supposed, and its prognosis is less favourable than that of the acute form. The animals become very emaciated and anæmic, frequently falling down from sheer weakness, and death from exhaustion is not uncommon. The chronic cases, in which diarrhœa persists for weeks and months, are generally incurable ; because they usually have their origin in lesions of the intestinal mucous membrane, as, for instance, atrophy of the epithelium with hypertrophy of the connective tissue of the mucous membrane, tubercular deposits and new growths.

Treatment.—Purely dietetic treatment suffices in many cases and may consist in depriving the patient of food and drink for some time, and we should above all things try to prevent the immoderate consumption of water. We have obtained a cure in many cases, merely by a change of food, especially when the animal has been fed on dog-biscuits, which are frequently injurious. The best food is raw meat given often and in small quantities. In cases of impaction of the stomach and in those of gastric catarrh caused by the presence of harmful food in the stomach, it is best to empty the stomach as soon as possible by an emetic. This procedure cures the gastric catarrh and prevents the occurrence of intestinal catarrh, which might otherwise follow as a consequence of the further advance of the irritating ingesta. In selecting an emetic, we should choose one which is least liable to irritate the stomach, and of these chloride of apomorphine is the best. It should be

given subcutaneously in doses of $\frac{1}{20}$ gr., to $\frac{1}{12}$ gr., to $\frac{1}{8}$ gr. to dogs and $\frac{1}{3}$ gr. to $\frac{3}{4}$ gr. to cats (1.100 C.C. of water, 1 C.C. of the solution to equal $\frac{1}{65}$ gr. apomorphine) or ($\frac{1}{4}$ fluid drachm to 4 fluid ozs. water; $\frac{1}{4}$ drachm to equal $\frac{1}{65}$ gr. apomorphine). Other emetics are: white hellebore ($\frac{3}{4}$ gr. to 3 grs.); ipecacuanha (8 grs. to 30 grs.); tartar emetic (1$\frac{3}{4}$ grs. to 5 grs., with plenty of water). Delicate lap dogs and fancy dogs should be given wine of antimony (German pharmacopœia), or of ipecacuanha—1 teaspoonful to 1 tablespoonful.

If there be loss of appetite, we may give hydrochloric acid (a teaspoonful to a tablespoonful of a mixture of 85 minims in 8 oz. of water); pepsin (hydrochloric acid and pepsin, up to 85 mins. in 9 ozs. water); the bitters (as, tincture of gentian 2$\frac{1}{2}$ drs., hydrochloric acid 1$\frac{1}{4}$ drs., water 8 ozs., 1 teaspoonful to 1 tablespoonful); tincture or infusion of rhubarb; pepsin-, chinchona,- or condurango-wine.

For diarrhœa we may at first prescribe a diet of dry food, toast water, rice water, barley water, and hydropathic packing of the abdomen; or we may give a teaspoonful to a tablespoonful of port wine. Later on, we may use laudanum or Dover's powder. Tannin, or its newer congeners tannoform, tannalbin and tannopin, may be tried. Schindelka recommends tannopin in doses of $\frac{3}{4}$ gr. to 30 grs. (1$\frac{1}{4}$ to 2$\frac{1}{2}$ drs. daily), and Shafer recommends tannoform in doses of 15$\frac{1}{2}$ grs. to 30 grs. (75 grs. daily) as being very efficacious in diarrhœa in dogs. In obstinate cases it may be necessary to resort to nitrate of silver in doses of $\frac{1}{8}$ to $\frac{5}{6}$ gr. with Armenian bole in a pill (argenti nitrici $\frac{3}{4}$ gr., boli 75 grs. M. ft. pil. XX.); Sig., one to two pills daily for a large dog; and, in very obstinate cases, nitrate of silver ($\frac{1}{10}$ to $\frac{3}{4}$ grain in a pill). Prior to this, we may employ enemas of solutions of alum, tannin or nitrate of silver (from 1 to 2 per cent.), which we have found to be very efficacious in some cases.

For constipation, which is seldom persistent in this disease, we may give calomel ($\frac{1}{2}$ to 1$\frac{1}{2}$ grains); for it not only acts as a purgative, but also disinfects the intestinal canal. We are rarely obliged to combat vomiting in cases of gastro-intestinal catarrh of carnivora. If it become troublesome, we may in the first instance give small pieces of ice, or ice-cream in the case of delicate dogs; or small doses of opium (1$\frac{1}{2}$ to 3 grains), tannin (1$\frac{1}{2}$ to 7 grains), bi-carbonate of soda (7 to 15 grains), bromide of potassium (15 to 50 grains) per rectum, oxynitrate of bismuth, creosote, creolin,

chloretone, hydrocyanic acid and tincture of iodine. For abnormal fermentation, we may give calomel, creolin (17 to 35 minims), and naphthaline (8 to 16 minims). After recovery, the mucous membrane of the stomach and intestines should be protected for some time from irritation by the avoidance of food which is difficult to digest.

CONSTIPATION IN DOGS.

Etiology.—Constipation occurs more frequently in dogs than in other domestic animals. Out of 70,000 sick dogs that were treated at the Berlin Canine Hospital during nine years, there were 1,275 (2 per cent.) cases of constipation, which is one in fifty. The disease, as a rule, takes the form of insufficient peristaltic action, or of mechanical obstruction, which hinder the passage and evacuation of fæces. On the other hand, many dogs seem to possess a predisposition to constipation (congenital muscular weakness of the bowel-walls ?). The chief causes are as follows :—

1. Food which is difficult of digestion or which is non-stimulating, especially too abundant feeding on bones; exclusive feeding on bread, dog-biscuits, pulse, porridge and dry food.

2. Insufficient exercise, particularly with dogs that are kept in-doors or chained up.

3. Old age, in which peristalsis becomes diminished, on account of atrophy of the intestinal mucous membrane and of the muscular coat of the intestines.

4. An existing chronic intestinal catarrh, which diminishes the sensitiveness of the mucous membrane by setting up atrophy in it, and by covering it with mucus, and which also decreases secretion of the intestinal juices, and reduces the activity of the reflex movements of the intestines.

5. Mechanical obstacles which impede the passage of the intestinal contents, as we find in constipation brought on by foreign bodies and accumulations of dung (*koprostasis*), especially dung which is full of bones; stenosis of the intestine by compressing tumours, among which, swelling of the prostate gland is prominent; pressure on the intestine by discharged abscesses; dilatations and paralysis of the intestine and diverticula of the rectum; piles; engorgement of the anal glands, and obstruction from outside by sticky and felted hair (so-called "false constipation"); constriction of the anus and rectum; perineal

abscesses (which, in a case observed by Baldi, contained numerous bot-larvæ !).

6. Constipation is also a *symptom* of various febrile diseases, in which the secretion of the intestinal juices becomes diminished ; of all chronic debilitating maladies, which bring on relaxation and mal-nutrition of the intestinal wall ; of acute gastro-intestinal catarrh after previous diarrhœa and after the too frequent administration of laxatives ; of jaundice caused by absence of bile, which should stimulate the peristaltic movements of the intestines ; of peritonitis and inflammation of the intestine due to œdematous infiltration and paralysis of the intestinal wall ; of spinal paralysis and cerebral disease, in which the constipation is a manifestation of disturbed intestinal innervation ; of diseases of the diaphragm and intestinal wall in consequence of diminished activity of abdominal pressure ; of muscular rheumatism, especially of lumbago ; of increased lactation, etc.

Symptoms.—Colic is less liable to result from constipation in dogs than in horses. The chief symptom is the more or less complete suppression of defecation. The patient makes frequent, violent, and occasionally painful efforts to evacuate, and is either unsuccessful, or passes small, dry, hard, single balls of excrement, which often contain undigested pieces of bones, which are usually of a mortar-like consistency, give off a penetrating and offensive smell, and are sometimes covered with mucus and blood. The appetite is at first decreased, and, later on, is suppressed for days and even weeks. Peristalsis is also in abeyance, and the abdomen is sometimes tense and tucked up ; at other times, tympanitic. On palpation of the posterior part of the body, which may be rendered very difficult on account of the tension of the abdominal wall, we usually find, at the entrance into the pelvic cavity, a hard, nodular or sausage-shaped mass of fæces (a so-called " fæcal tumour "). This fæcal cord may reach as far forward as the ensiform cartilage, and its palpation generally causes pain. The parts around the anus are often inflamed, swollen and soiled with yellowish, gritty excrement. In cases of so-called " false constipation," the tenacious, semi-solid stools form, with the hair around the anus, a stiff mass that renders defecation impossible. On exploration of the rectum, which is usually very painful, we find the temperature raised, the mucous membrane dry and swollen, and the rectum often

filled with excrement chiefly composed of bony particles. In other cases, we fail to reach the accumulated fæcal masses, or succeed only in touching them with the end of the finger. Sometimes the excrement which had collected in front of the "fæcal tumour," burrows its way through it, and may appear in the form of fœtid diarrhœa intermixed with a good deal of gas. Vomiting often occurs in cases of complete intestinal obstruction.

Fever is either entirely absent, or of a very mild type in cases of long-continued constipation. Depression gradually increases; colic occasionally appears, and exercise is avoided. Male dogs now and then micturate without raising a hind leg. The peculiar manner in which the tail is stretched out, or considerably bent at the root, is very characteristic, and is often sufficient for diagnosis.

Course.—The disease generally lasts from a few days to a month or six weeks. In more prolonged cases, the action of the accumulated fæces on the intestinal mucous membrane gives rise to symptoms of diphtheritic intestinal inflammation, intestinal necrosis, septicæmia and perforating peritonitis. Cicatricial strictures of the mucous membrane and diverticulæ of the intestinal canal are sequelæ which may cause recurrent attacks of constipation. The average duration is from a week to a fortnight, when the disease generally ends in recovery. With regard to differential diagnosis, it may happen that failure to pass fæces, as a consequence of long fasting, may be mistaken for constipation. Confusion between "fæcal tumour" and true new-growths may also occur, although the differentiation will be easy, if we pay attention to the form, hardness and position of the swelling, to the history of the case, and to the further course of the disease. In difficult cases, if the obstruction is reduced by the employment of purgatives and clysters, any suspicion of a new growth will be removed.

Treatment.—Treatment is usually dietetic, mechanical and medicinal. In very rare cases, such as those of abscesses, swelling of the prostate gland and foreign bodies in the intestine, surgical aid may be required.

As regards *diet*, bulky and indigestible food (bread, bones, dog-biscuits, potatoes, etc.), should be avoided. At first, we may keep the patient fasting, and allow him only fresh cold

water. In long standing cases, we should feed him only on milk, salt meat broth and, possibly, lean meat free from bones. At the same time, we should try to stimulate peristalsis by frequent exercise.

Among *mechanical* means, ordinary enemas or injections of large quantities of water by means of an india-rubber pump are very effective. Pure warm water is best for softening and washing out the accumulated fæces. Clysters of cold water are to be preferred only in cases of intestinal paresis. We may try injections of from 7 to 14 oz. of olive oil, three times daily. Glycerine is generally useless for this purpose. We should also try to remove the constipation by frequent massage, namely, by carefully pressing and kneading the obstructing fæcal ball from the outside, and by exploring the rectum with the finger, forceps or curette. These manipulations demand the expenditure of much time, patience and trouble.

These mechanical means may be supplemented by the use of purgatives. Castor oil (⅔ oz. to 1⅔ oz. in an emulsion) or calomel (¾ gr. to 1¾ grs., as a powder with sugar) are generally given. In very obstinate cases, supposing that the intestine is in no way inflamed, we may try from 1 to 5 drops of croton oil, with gum arabic and castor oil. We should bear in mind that the strongest cathartics will be unable to remove the ball of fæces, if it be wedged in the intestinal canal, or if the intestinal wall be paralysed. After recovery, the dog should be appropriately fed, so as to avoid a relapse.

CONSTIPATION IN CATS.

Constipation is rare in cats and, when it occurs, is generally caused by foreign bodies, diverticula and new-growths. Von Puntigam has described a case of feline constipation caused by a recto-vaginal fistula. The treatment consists of enemas and the administration of calomel.

FOREIGN BODIES SWALLOWED BY DOGS.

Etiology.—As in cattle, foreign bodies cause in dogs, and less frequently in cats, peculiar morbid conditions which may not be diagnosed during life, and are often mistaken for symptoms of rabies. In some cases foreign bodies cause hæmorrhages and perforation of the stomach and intestines, as well as conditions requiring a purely surgical treatment.

Such are fistulæ caused by the perforation of the gastric or intestinal wall. Other affections produced by these bodies will be discussed later on under the heading of "Peritonitis due to Perforation." A third variety of these accidents gives rise to dangerous gastric symptoms by impaction of the stomach or intestines, or by a more or less complete obstruction of the intestinal lumen, and by severe hæmorrhagic and necrotic inflammation of the mucous membrane.

Foreign bodies, which cause the accidents in question, are usually large pieces of bone, stones, metal and glass balls, coins, buttons, corks, pegtops, sponges, chestnuts, nuts, balls used in play, reels of cotton, pieces of wood, cord, children's shoes, etc. They are usually swallowed accidentally when fetching and carrying, or when playing; but may also be swallowed unintentionally during mastication. For instance, while a dog was fetching a grape-shot ball which weighed more than ½ lb., he swallowed it. We once treated a dog which used to fetch coins, and swallowed, on three different occasions, a two-shilling piece. It is a strange fact that if needles, which are swallowed, do not perforate the oral cavity or pharynx, they rarely cause any harm. As an experiment, Zoja made cats swallow needles, some of them with the points foremost. Out of 127 swallowed needles, only two perforated the alimentary canal; one just above the pylorus; the other in the rectum. Of the remaining 125 needles, some were passed out with the dung in from 4 to 140 hours; and others were found in the large intestine without having caused injury to the stomach or intestines of the cats which had swallowed them, and which, in order to make this investigation, were killed. Similar instances have been seen in mankind. For instance, a sempstress swallowed 1,000 needles without injury.

Symptoms.—These are generally of a vague nature. At first, as long as the bodies lie free in the stomach, no morbid disturbance is usually noticeable. For instance, Nichoux saw a dog in whose stomach a four-shilling piece and a penny remained without any inconvenience for 12 years, at the end of which time the animal accidentally died, and these coins were found in his stomach. In other cases, acute gastric disturbance is at first set up, and, later on, if the foreign body remain for a long time in the intestinal canal, it may produce a chronic gastro-intestinal affection. If the swallowed body become

impacted in the stomach or intestines in such a manner as to more or less completely occlude the passage, the symptoms naturally are more grave. There is usually no fever at first, although the dog becomes peculiarly depressed in spirits, lies down a good deal, eats little, and drinks often and copiously. These symptoms become complicated with vomiting, which frequently takes place with a certain regularity as to the time after the consumption of food or water. It is often performed with extraordinary violence and persistence, in which case, mucous, bilious and even fæcal matters are thrown up. Well-marked jaundice is not uncommonly observed. Defecation is more or less completely suspended. Fever is also a symptom; in some cases the temperature may remain for a comparatively long time only slightly above normal; in others it rises very quickly. Appetite is entirely suppressed, and emaciation rapidly sets in. Symptoms similar to those of rabies are sometimes seen. The patient becomes depressed; tries to hide himself; is exceptionally irritable and excited; has convulsions and epileptic spasms; runs about aimlessly; and may manifest a strong desire to bite and a mania for destruction. Towards the end, rapidly increasing debility sets in; the expression of the face becomes plaintive; there is extreme and very rapid emaciation; the temperature rises; the pulse becomes weak and slight; the patient falls into a comatose condition and finally dies.

Diagnosis.—The history of the case and palpation of the posterior part of the body are more important for diagnosis than the symptoms we have described. Palpation should never be omitted, because it enables us in the majority of cases to determine the presence of a foreign body in the stomach or intestines. The exploration of the stomach is best effected by making the animal stand on his hind legs, and pressing on the posterior abdominal wall behind the ensiform cartilage and that of the intestines, by making the patient stand on all four legs, or by placing him on his back, and pressing on the abdominal wall.

Should an apparatus be available, the exact position of metal foreign bodies may be determined by means of the Röntgen rays (Mollereau and others).

Duration.—The duration of the disease is very variable, and may end at any time from a few days to several months,

according to the degree of obstruction set up. We may occasionally observe a series of improvements and relapses, according to the course taken by the foreign body. Needles, in particular, show an inclination, after having perforated the wall of the intestinal canal, to wander through the body and leave it, by the formation of superficial abscesses on the ribs, flanks, tail, etc.

Post-Mortem Conditions.—If the foreign body be impacted in the pylorus, the right half of the stomach will be distended, and its muscles more or less hypertrophied, according to the duration of the disease; in fact, we have found them two-fifths of an inch thick in a medium sized dog. In such cases, the intestines will be nearly, if not quite, empty. If the foreign body has stuck fast in the small intestine, the portion of the intestine which is in front of it will be dilated and hypertrophied, and the rear portion narrowed and empty. At the site of the impaction the mucous membrane will be inflamed or will have a necrotic scab resembling tinder in appearance, which changes may sympathetically appear in other membranes, including the peritoneum. The way by which the body has travelled is marked by ulcers, scabs, extravasations of blood, pigmentation, etc. Sometimes the arrest of the body is caused, not so much by its large size, as by the irregularity of its surface (as in corks), which leads to paresis of the intestinal muscles, and the obstruction, therefore, remains at a certain spot.

Therapeutics.—Treatment should commence with the administration of emetics or purgatives. As long as foreign bodies, such as coins and stones, remain in the stomach, they may often be promptly removed by an emetic (apomorphine subcutaneously, for instance); and, if in the intestine, by a cathartic, unless they are impacted, in which case, gastrotomy or enterotomy will be needed. These operations, which have been successfully performed by many veterinary surgeons, should be more frequently practised than has hitherto been done, because they entail but little danger on dogs when properly carried out. The prognosis of laparotomy is naturally unfavourable when once the foreign body has set up necrotic inflammation in the intestine.

The swallowing of foreign bodies by cats.—The objects swallowed are usually needles, balls of cotton, or pieces of cork,

In resorting to operations the use of carbolic acid should be avoided, as it is poisonous to cats. Hobday describes such a case.

GASTRO-INTESTINAL CATARRH OF PIGS.

Etiology.—The causes of gastro-intestinal catarrh in pigs are the same as in dogs. This disease sometimes occurs epizoötically (infectious gastritis), mostly during wet seasons, and among pigs at grass; and originates from chill, inordinate consumption of snails and worms, or by infection.

Symptoms.—The chief symptoms are: impaired appetite, vomiting, restlessness resembling that due to colic, constipation, diarrhœa, fever, coldness of the extremities and especially of the ears, dry and hot condition of the snout, congestion of the visible mucous membranes, digging and hiding in the litter and drooping of the tail. The same symptoms are to be seen in "infectious gastritis," which usually runs a slow, chronic course, and often ends in death from gradually increasing prostration and emaciation. Cases of epizoötic gastro-intestinal catarrh among swine have been described by Marks, Aronsohn and Esser. In the first case (Marks) the symptoms were loss of appetite, fever up to 105.4° F., constipation at first and watery diarrhœa later on. All the swine recovered in eight days. In the second case, Aronsohn observed that all the pigs upon several estates were attacked by the following symptoms: thin, slimy diarrhœa, lassitude, stiff or staggering gait, and in isolated cases, vomiting. The duration of the disease was about eight days and the mortality one to two per cent. The *post-mortem* appearances in most cases were those of simple catarrhal (in two cases hæmorrhagic) gastritis and enteritis. In the third case (Esser) the disease was brought to a farm by some newly-imported breeding sows. The chief symptoms were loss of appetite, and diarrhœa. These gave way to a purely dietetic treatment and disappeared at the end of eight days.

Therapeutics.—At first the treatment is somewhat similar to that in dogs, with the exception that the doses are larger, and that the method of administration is a different one. Drenches should be avoided, because they are difficult to give, and are liable to set up drenching pneumonia. The medicine should usually be administered in the food; or as an

electuary, enema or subcutaneous injection. In all cases of impaction of the stomach, or of spoiled, bad food having been eaten, treatment should be commenced with an emetic. We generally use veratrine subcutaneously (½ grain to 1½ drs. of spirit brings on vomiting within an hour). Hydrochlorate of apomorphine is ineffective as an emetic in pigs. Ipecacuanha and white hellebore powder are not always easy to give by the mouth; but a decoction or infusion of hellebore (15 grains in 2 oz. of water) may be given as an enema. In constipation, we may give calomel, but in much larger doses than to dogs (15 to 60 grains), sulphate of magnesia, Carlsbad salt, enemas of soap and water and copious water injections. For diarrhœa we may try 15 to 30 grains of opium, or tannin, tannalbin, tannopin, tannoform, etc., may be employed. Easily digestible and cooling food, such as sour milk and gruel, should be provided.

The swallowing of foreign bodies by swine.—As in the case of cattle and dogs, foreign bodies (knitting needles, sausage skins, sand) are often found in the stomach and bowels of pigs. As a general rule, these are discovered only after slaughter. The symptoms during life are loss of appetite, vomiting, constipation, emaciation, jaundice (arising from the occlusion of the gall ducts by sand), etc.

GASTRO-INTESTINAL CATARRH IN BIRDS.

(Diarrhœa and Constipation.)

Simple Gastro-intestinal Catarrh and Diarrhœa of Birds.—This disease must be clearly distinguished from infective diarrhœa (fowl cholera), disease caused by gregarines (gregarinosis), and severe inflammation of the intestine, as in cases of poisoning. It is found most frequently in young birds of fancy imported breeds, and especially during spring, while the young birds are moulting. As in other domestic animals, its causes are errors of diet and chill. Diarrhœa occurs secondarily from entozoa, tuberculosis, etc. The chief *symptoms* are: Diminished appetite; ruffled plumage; depression of spirits; slow evacuation of the crop; the passage, at first, of soft, white, slimy fæces, which later on become watery, and of a greenish white colour, and soil and stick together the feathers near the arcus, eroding the inner and outer surface of the cloaca; increased thirst, emaciation and weakness; and finally, if the diarrhœa does not stop, death ensues from exhaustion or hæmorrhagic intestinal inflammation. On *post-*

mortem examination, we find signs of desquamative intestinal catarrh, congestion, swelling, degeneration of the epithelium of the mucous membrane or inflammation of the intestine. At first the *treatment* should be dietetic. Above all things, the birds should be kept warm, and the fowl-house clean. The food should be easy of digestion, and therefore should be boiled. We may combat the diarrhœa by giving parched barley, rice water, old biscuits, hemp seed, blue poppy seed, and in the case of parrots, small pieces of chocolate, bread moistened with spirits, a teaspoonful of port wine, or bread or grain soaked in port wine. Zürn recommends oatmeal (a tablespoonful to a pint of water) or linseed tea (1 to 20 of water). As regards styptics, we generally use a watery solution of sulphate of iron (1 per cent. in drinking water). Opium is also very effective with birds, and may be given in the form of laudanum (fowls and parrots, 1–5 minims; pigeons, ¾ minim). In very obstinate cases, Zürn gives fowls one or two teaspoonfuls of a 1 per cent. solution of nitrate of silver daily. For simple digestive disorders, he gives minced onions or garlic, and a few pepper-corns made up with butter into pills. An infusion of peppermint or calamus (2 in 60) may be given; fowls 1 tablespoonful, pigeons 1 teaspoonful, 3 times a day.

So-called "Atrophy" in birds.—According to Schindelka, birds, and especially those eating maize, are subject to an affection which manifests itself in an abnormal hunger, gradual emaciation and very thin diarrhœa. The cause is to be sought, not in tuberculosis, but in improper feeding.

Constipation of Birds may be caused by previous diarrhœa, entozoa, stones, feathers and other foreign bodies in the stomach and intestines; dry dung in the cloaca; digestive inactivity of the intestinal canal, etc. *Treatment* consists in giving green food, slimy and oily substances, cold water enemas; and with small singing birds, in passing into the rectum a bulb-pointed probe smeared with castor oil, and scooping out the fæces which have collected in the cloaca. The best aperients are castor oil (2 tablespoonsful for a strong hen), senna leaves (15 to 20 grains), rhubarb (3 to 7 grains, made into pills with honey), and calomel (¾ to 1½ grain). To small cage birds we give ½ per cent. watery solution of artificial Carlsbad salt as drinking water.

COLIC IN HORSES.

Definition.—The term colic (enteralgia) is usually applied

to various morbid conditions of the stomach and intestines, of which pain is the chief symptom, and which are generally associated with suppression of intestinal peristalsis. Hence, it does not signify any special disease. Colic, like broken-wind, dropsy, jaundice, etc., has a wide collective meaning. Besides "true colic" (pain in the stomach and intestines), we have so-called "false colic," arising from painful affections of the kidneys, bladder, liver, uterus and other organs that lie in the abdominal cavity, and also certain general affections, as for instance, petechial fever and anthrax, which show secondary symptoms of colic.

The name "Colic" has kept its place in text-books of special pathology because of its long continued employment. In the case of the horse as compared with other domestic animals, colic is a peculiarly characteristic and unique symptom. For the pathological conditions of stomach and intestine which give rise to colic in the horse are, in the case of the other domestic animals, frequently unattended by any such clear manifestation of pain. It is very difficult in most cases to recognise the nature of the particular affection to which the symptom is due, and for this reason, especially at the beginning of an attack, diagnosis is frequently confined to the establishment of colic as the chief symptom.

History.—Colic is one of the oldest known equine diseases. Its literature can be traced back to Columella (1st century after Christ), who described three kinds of colic. Eumelus (3rd century) alluded to saltpetre as a remedy for colic. In the 4th and 5th centuries Vegetius, Apsyrtus, and Hierocles wrote about the symptoms of colic with a fair amount of accuracy. Vegetius, in particular, describes various kinds of colic, such as *dolor ventris, dolor coli, et dolor vesicæ et ileus.* In the 13th century Jordanus Ruffus distinguished between flatulent colic and colic caused by over-feeding. In the 16th century colic is fully described, especially in the writings of Vegetius Renatus and Joannes Ruellius. The works of Ehrmann, Sander and Weber appeared in the 18th century, and those of Ribbe and von Tennecker in the first half of the 19th. Among recent writers on this subject we may mention the names of von Ullrich, Legrain, Bollinger and Friedberger.

Statistics.—Colic is the most important of all equine internal diseases. Out of about 70,000 cases of illness, which occur yearly among the horses of the Prussian army, about

3,000 are those of colic. Bollinger computes that out of a total number of 400,000 horses in Bavaria, about 40,000 are attacked yearly by colic; and states that the mortality is about 12 per cent. (1,625 fatal cases in 12,857). Among the 26,253 cases of colic which were reported to us by different veterinary colleges during ten years there were 2,832 fatal cases, which give a percentage of about 11. In the Prussian army the average mortality is 12 per cent. According to Bollinger, 40 per cent. of deaths occur from colic.

In the hospitals of veterinary colleges the mortality from colic has been, on an average, about 14 per cent.

The figures are as follows :—

Place.	Number of cases.	Number of years.	Deaths.	Percentage.
Vienna	16,097	25	1,643	10·2
Hanover	1,200	10	134	11·1
Munich	1,606	10	201	12·5
Dresden	636	16	86	13·6
Stuttgart	1,088	19	184	17·0
Berlin	1,815	10	354	19·0

In the Prussian army in 1880-1900 there was an average mortality among about 60,000 horses suffering from colic of 12 per cent. In 1881-1900 the results were as follows :—

Year.	Number of Sick Horses.	Deaths.	Percentage.
1881	2,145	302	14
1882	2,368	324	13
1883	2,578	218	8
1884	2,546	249	9
1885	2,719	306	11
1886	2,515	257	10
1887	2,147	249	12
1888	2,642	261	10
1889	3,108	407	13
1890	2,980	447	13
1891	2,951	209	14
1892	3,353	427	13
1893	3,509	443	13
1894	3,661	496	13
1895	3,509	496	14
1896	3,413	449	13
1897	3,519	478	12
1898	3,482	463	13
1899	3,082	450	14
1900	3,746	528	14

General causes of Colic.—1. *Predisposing factors.*—Horses, by reason of the structure of their stomachs and intestines, are more liable to colic than any of the other domestic animals. We must here bear in mind that the equine stomach is very small, and, even when filled, does not reach the abdominal wall; the œsophagus enters the stomach obliquely, and is furnished with a regular sphincter muscle; and the stomach possesses a well-developed cul-de-sac. These peculiarities as a rule prevent vomiting and the removal of solid, liquid and gaseous substances by the mouth. The great mesentery is so long, and the large intestine so voluminous and so free to move about, that it can readily become displaced or impacted with food. Also the terminal ends of the intestinal nerves, like those of the peritoneal nerves, are so sensitive to external influences, that colic may be set up without any apparent cause. And finally, aneurism of the anterior mesenteric artery, which is found in most horses, is a predisposing cause of colic, in consequence of thrombosis and embolism of the intestinal arteries.

2. *Over-feeding*, especially when the quantity consumed is out of proportion to the amount of work, as often occurs on Sundays and holidays. The effect of over-feeding is intensified by the fact of the fodder being difficult to digest, as in the case of straw, chaff that has been cut too short, new corn, new hay, raw potatoes, maize, rye, beans, peas ; by sudden change of food ; by working the animal directly after feeding ; and by the food being imperfectly masticated, owing to defective teeth.

3. *Spoilt food*, such as : hay or grass which has become muddy, sandy or diseased, or has been trodden down ; damp, fungus-ridden straw ; mouldy barley straw ; dirty, rotting, mouldy or spoilt litter ; mouldy, fusty oats ; mouldy bread ; distillers' wash ; dirty, cloudy, muddy or highly mineralised water ; the eating of spoilt or damp litter is the most frequent cause of indigestion colic in cavalry horses.

4. *Formation of gas* in the stomach, produced by eating young or decayed clover, especially red clover, lucerne and sainfoin, decaying grasses, rank vegetation, new grains, potato leaves, etc. Fermentation is often set up by drinking water soon after feeding. The swallowing of large quantities of air when wind-sucking may also lead to colic.

5. *Accumulation of food in the intestines*, as may occur from too long continued rest ; feeding on too much innutritious

food, such as straw or husks, or on too dry food ; from the treatment of paralysis of the shoulder by combined morphium and atropine injections ; and from relaxation, dilatation or paralysis of the intestines.

6. *Chill.*—This may result from a sudden lowering of the temperature of the surface of the body in cold or wet weather ; a sudden change from dry weather with an east wind to damp weather (at such times colic is almost enzootic) ; from changing the coat ; from very cold, frosted or frozen fodder or from too cold drinking water. Among army horses, a large percentage of the cases of colic are due to chill.

7. *The formation of calculi and concretions, and the accumulation of large quantities of sand and earth in the stomach and intestines*, as often happens in campaigns and during military manœuvres, and with horses at grass. Concretions are sometimes formed in the intestines of the horses of millers and bakers by long continued feeding on bran and sharps, both of which contain a large amount of phosphate of magnesia. Gall stones are rarely a cause of colic.

8. Intestinal worms, especially collections of *ascaris lumbricoides*.

9. Intestinal stenosis from compression, displacement, volvulus, invagination and incarceration of the intestine.

10. Long fasting (hunger colic), which occurs chiefly among military horses in war time and during manœuvres, as well as after being ridden or driven long distances.

11. Rolling produces colic in rare instances by displacement of the intestines. Thus, Köhne saw a case of volvulus caused by a horse being rolled on his back when cast for an operation. It is a well-known fact that recent twists of the guts are found at slaughter houses in pigs and calves which have been rolled about a good deal during unloading. On account of these exceptional cases, it would be unreasonable to prevent the rolling of a horse which was suffering from colic. Experience shows that horses at liberty roll with impunity, and that recent volvulus has often been cured by rolling.

12. Colic is met with in certain diseases of the intestinal tissues, such as catarrh, inflammation, croup, ulcers and poisoning.

Symptoms.—The most prominent symptom is pain, which may come on suddenly or gradually. If in harness or saddle, the horse goes " short," gives way in his hind-quarters, stops,

becomes restless, paws the ground and stamps, shakes himself, and can be prevented only with difficulty from lying down. In the stable the true pains are sometimes preceded by depression of spirits, loss of appetite and hanging back from the manger. Later on, he often totters, groans, kicks with the hind legs at the abdomen, looks round at his hind-quarters, whisks his tail, stands over at the knees, arches his back, stretches out his head and neck, and places his hind feet under his body. As a rule, the animal lies down very carefully and slowly, or tries to lie down, but sometimes may throw himself recklessly on the ground. When down, he may lie quietly, or may throw himself about, roll and kick; or he may remain quietly on his back, with the legs drawn towards the body, for a long time. The pains may temporarily disappear, and the horse get up and begin to feed again, but the state of uneasiness will soon return.

The bodily temperature is unequally distributed, and the ears and extremities are usually cold. Sweat often breaks out, especially at the base of the ears, on the sides of the neck and ribs, belly and flanks, which parts are sometimes hot, at other times cold, and occasionally the sweat drops on the ground. The visible mucous membranes are usually red, or may be discoloured in various ways. The oral cavity is very dry and hot, and its mucous membrane sometimes affected with catarrh; or it may be very cold and pale, with a washed-out appearance. The abdomen is tense, and may be more or less distended with gas, but it is tucked up only on rare occasions. Usually peristalsis is greatly diminished or entirely suppressed, and very faint, if any, intestinal murmurs are heard on auscultation; but in exceptional cases loud metallic sounds are heard. Defecation is delayed and the fæces, as a rule, are dry, hard, formed into small balls, covered with mucus and occasionally bloody. In many cases, only a few balls of dung are pressed out by great straining. Sometimes the fæces are very soft and even watery, are of an acid smell, contain undigested particles of food, and flatus frequently escapes. Occasionally necrotic and desquamated pieces of the intestine are found among the fæces.

On exploring the rectum, we may find it completely filled with dung, quite empty, or containing only a few balls of fæces. The mucous membrane is hot and swollen; the neighbouring portions of the intestine are often firm and tense to the touch, in consequence of their being filled with excrement or gases, and the bladder may be excessively filled. The lumen of the

rectum may be greatly dilated, or so much narrowed, that we may be unable to pass even one finger through the constricted part. Examination of the inguinal rings in stallions may reveal a strangulated hernia. When the colon is abnormally distended, we may feel one of its longitudinal bands in the form of a tensely stretched cord in the abdominal cavity. In rare cases we may find a calculus in the rectum. Micturition is usually delayed, although the animal often stretches himself out, draws his penis and tries to stale, but as a rule is able to pass only a few drops of urine, which may be clear in colour, or of such a rich tint that it may appear to contain hæmoglobin. It may be of an alkaline, neutral or acid reaction and now and then contains albumen.

The breathing is generally laboured and often very difficult. The pulse at first suffers no great change, but later on its rate increases to 50 or 60 beats per minute and, in extremely grave cases, to 100 or more. The heart-beat becomes stronger, and the cardiac sounds are often very loud. The temperature is normal at the beginning of the attack, but may rise to a high degree during the further progress of the disease, although the fever may abate to a great extent shortly before death. The animal's mental faculties are more or less affected, and any bad habits which he may have, like crib-biting, are discontinued.

In graver forms of colic more severe symptoms are seen. The animal assumes peculiar attitudes; for instance, he kneels down, while keeping his hind-quarters raised; lies on his sternum with his fore-legs stretched out (in a position similar to that of the Sphinx); sits like a dog, namely, on his hind-quarters with the fore-quarters raised. The animal eructates, stretches himself out, retches and may vomit, in which case the vomited matter is often discharged through the nostrils. The pain may increase to such a degree that the animal becomes maddened, rears, snorts, groans, screams, may even attack men, bite the manger or parts of his own body, grind his teeth, or may press forward as if stunned; circle round and round always in the same direction; make nodding, jerking and pendulous movements of the head; lean senseless and stupefied against the wall; tremble, become convulsed and covered with a cold and profuse sweat. The pulse is thready, intermittent or imperceptible; the heart-beat tumultuous; the expression haggard; the pupils dilated; the lower lip pendant; the rectum gaping and sometimes prolapsed, and a brownish fluid flows

from the anus. Finally, the animal staggers, falls on the ground, becomes convulsed and dies. Sometimes he neighs loudly before dying and may occasionally drink a large quantity of water. At other times the pain suddenly ceases, and coma speedily follows.

Course and Duration.—In the large majority of cases the course is short and acute. Occasionally the symptoms of colic disappear in a few minutes, but usually they continue for some hours; their longer persistence than thirty-six hours is a very unfavourable sign. Colic may, however, take a subacute or chronic course, in which case it may last for a fortnight, and with intermissions for several weeks, especially when the malady is due to constipation. In cases of this description the attacks alternate with painless periods. Of the complications associated with colic, those which are of most frequent occurrence result from decubitus. Sores appear upon the projecting portions of the body where the skin lies close to the bone, such as the orbital arch, the zygomatic arch, the posterior edge of the jaw and the anterior iliac spine. Other injuries of the most varied description, caused by the animal throwing itself down and rolling about, may occur, such are strains, sprains and fractures. Internal injuries are also by no means uncommon. Pneumonia, due to the presence of foreign matter, frequently follows upon unskilful drenching. This is especially likely to be the case if the head is held too high or if the liquid enters by the nasal passages. Thus, vomiting occasionally gives rise to pneumonia, owing to the inspiration by the animal of the voided food stuff. Symptoms of stomatitis and of pharyngitis, both slight and severe, may be found after the administration of too hot drenches, or of strong purgatives, such as croton oil and tartar emetic, when insufficiently diluted. Similar changes may be seen in the vaginal mucous membrane of mares after the so-called "salt and pepper" treatment, as well as in the rectal mucous membranes after careless handling when "clearing out" the rectum. In the latter case, the condition may lead to perforation of the rectum.

Prognosis.—In no case of colic can the result be anticipated with certainty. Cases which are apparently slight may, owing to intestinal displacement, terminate fatally. With the same case, prognosis may vary from minute to minute. Hence the prognosis of colic should always be made with reserve.

Signs of improvement are:—Abatement of pain; a more uniform distribution of bodily temperature; cessation of sweating; improved general condition; improvement in the condition of the pulse; return of the peristaltic sounds; evacuation of fæces and flatus; return of appetite, desire for water, fall in temperature, normal pulse, etc.

The following are *unfavourable signs*:—Symptoms suggestive of gastro-intestinal inflammation, peritonitis and rupture of the stomach, intestine or diaphragm, which symptoms are generally as follows: hard, small and very frequent pulse; very high or sub-normal temperature; pain becoming suddenly more acute or ceasing altogether; cold sweats; great coldness of the extremities; persistent constipation; complete suppression of peristalsis; great distension of the abdomen; dyspnœa; vomiting; staring and glassy expression of the eyes, etc. For further symptoms see under special colic headings.

General Differential Diagnosis.—True colics, which are caused by pathological conditions of the stomach and intestines, may be confounded with a large number of painful affections in other organs, such as:—

1. *Rheumatic Hæmoglobinuria*, the symptoms of which are often very similar to true colic, as, for instance, its sudden appearance after a chill, manifestations of intense pain, cessation of peristalsis, suppression of defecation and micturition, violent outbreaks of sweat, dyspnœa and derangement of circulation. It is probable that, on this account, rheumatic hæmoglobinuria has been frequently mistaken for colic. Close examination will, however, give us ample data for their differentiation. In true colic there is no paralysis of the hind limbs, pain nor swelling of their muscles, which are characteristic symptoms of the other disease. The urine also contains neither hæmoglobin nor dissolved blood. In true colic the animal often lies down and gets up again; but in rheumatic hæmoglobinæmia he is generally unable to rise.

2. *Intestinal catarrh and enteritis*.—Of the morbid conditions affecting the intestinal tract, catarrh, inflammation and ulceration are the most frequent causes of colic. "Indigestion colic" and "inflammation colic" are terms in common use. But as catarrhal, inflammatory and ulcerous conditions of the gastro-intestinal mucous membrane constitute distinct lesions and must be treated as such, it is necessary to distinguish between

the symptomatic colic of these conditions and the various forms of true colic. This is not always an easy task, but the following points should be borne in mind. In intestinal catarrh, the colic is a secondary symptom and soon gives way to treatment; diarrhœa is usually present, and the attack is of a slighter nature and lasts longer than in true colic. In intestinal inflammation, the previous history is of the utmost importance in determining whether the condition is due to irritation of the bowel by sharp substances or by poisons (poison colic). Severe, continuous and frequently sanguineous diarrhœa with high fever following upon extreme constipation are indicative rather of an inflammatory condition of the mucous membrane than of colic. Ulcers in the stomach and intestines are difficult of diagnosis in any case, are frequently accompanied by hæmorrhage and take a chronic course. True colic, on the other hand, constitutes an essentially acute condition.

3. *Labour pains.*—Certain symptoms resemble those of colic. The animal paws the ground, moves about restlessly, groans, and frequently lies down. But a careful examination of the uterus, vulva, teat and flanks, together with the previous history and the fact that labour pains are periodical in their nature, alternating with painless intervals, should render the distinction an easy one.

4. *Retention of urine* from paralysis or obstruction, which is often mistaken for ordinary colic by lay observers or the inexperienced, because the pressure exerted upon the bladder by the distended intestine excites the animal to make efforts to urinate, even when the bladder is empty. Examination of the bladder by the rectum, or the introduction of a catheter, will give the necessary information. Retention of urine, which is rare in horses, does not interfere with the activity of the digestive organs.

5. *Peritonitis.*—It is always difficult, and sometimes impossible, to distinguish between peritonitis and colic, especially when the latter has a rheumatic origin. In fact, peritonitis can be diagnosed in horses only in certain cases. It can be recognised with comparative ease when it is a secondary inflammation resulting from an injury to the peritoneum, whether accidental or the result of an operation, as after castration, laparotomy and herniotomy; or when it follows inflammation of the kidneys, bladder or uterus; the primary affection in such cases determining the cause of peritonitis. The peritonitis which may occur during colic can often be recognised by the height of the accom-

panying fever, the smallness and frequency of the pulse, and by rapidly increasing prostration.

6. *Strangulated hernia.*—Inguinal, umbilical, ventral, and scrotal hernia present a clinical aspect similiar to that of severe colic. A careful examination, especially of the scrotum and inguinal ring in stallions and geldings, will, however, reveal the true nature of the affection.

7. *Inflammation of the bladder.*—Ample material for diagnosis is afforded by the characteristic condition of the urine which contains epithelial cells, pus corpuscles, and triple phosphate crystals; by the sensitiveness of the bladder when pressure is exercised by way of the rectum; the frequent passage of urine in minute quantities; and the complete immunity of the gastro-intestinal canal.

8. *Metritis* may be distinguished from colic by the fact of a preceding birth, the usual presence of a discharge from the vagina, abnormal sensitiveness of the uterus and longer duration of the disease.

9. *Displacement of the Uterus* (torsion, prolapse, inversion and invagination). With internal and external examination only incomplete prolapse will present any difficulty in diagnosis.

10. *Nephritis.*—The symptom upon which diagnosis is chiefly based is the presence of certain matters in the urine. These are, red and white corpuscles, epithelial cells, casts and albumen. A microscopic examination of the urine should therefore suffice to establish a diagnosis. The symptoms of renal colic are much less severe than those of true colic and frequently manifest themselves only in increased sensitiveness over the lumbar region and in a stiff and strained gait.

11. *Inflammation of the Liver.*—Acute hepatitis, which is exceedingly rare in horses, manifests itself in slight restlessness, which is probably traceable to inflammatory irritation of the peritoneal covering of the liver. If no distinct icterus be present, it might be difficult to make a correct diagnosis.

12. Finally, various diseased conditions of the body may be mistaken for colic, if accompanied by acute pain; but careful examination will always prevent such errors. These conditions are: inflammation of the brain, pleuritis, pneumonia, petechial fever, influenza, painful surgical affections, especially inflammation of the hoofs or joints, and, in certain cases, also thrombosis of the iliac and femoral arteries.

General Anatomical Notes.—From *post-mortem* examination we find that the phenomena of colic vary greatly according to its cause. Volvulus of the colon and small intestine, as well as laceration of the stomach are most frequent, and occur uniformly in the ratio of one in five of all fatal cases.

Of 1,882 *post-mortem* examinations made in the Prussian Army during five years, 347 cases, or 18 per cent. of the whole, were found to be cases of stomach-laceration; 336, or 17 per cent., were volvulus of the colon; 331, or 17 per cent., were volvulus of the small intestine. Thus the above-mentioned three pathological changes were found in about half the entire number of cases. Next in frequency came gastro-intestinal inflammation and laceration of the colon (each 8 per cent.), also laceration of the diaphragm with prolapse of parts of the intestine (5 per cent.). Less frequent were laceration of the omentum or mesentery with confinement of portions of the intestine; in the Winslow's foramen, obstruction of the colon and volvulus of the cæcum (3 per cent.). Bowel-stones were rare, as also was compression of the small intestine by lipomata, laceration of the cæcum or of the rectum, invagination of the small intestine into itself or into the cæcum, diverticula of the intestines and obstruction of the small intestine (each from 0.5 to 1.5 per cent.). Volvulus of the rectum, invagination of the rectum into itself, worms, etc., occurred only in isolated cases as the cause of death.

With regard to these changes we find that in displacement (volvulus) of the intestines the twisted or knotted part of the bowel is generally very pale or anæmic, while the walls of the intestine, both in front of and behind the affected spot, are swollen and infiltrated with blood, the mucous membrane is dark red and tender, the contents of the gut are blood-coloured and the mesentery is gelatinous and charged with blood. Collections of foodstuff, stones and concretions all lead to catarrhal, hæmorrhagic and diphtheritic or necrotic inflammation of the mucous membrane. They chiefly occur at the junction of the ileum and cæcum, and at that of the colon and rectum, and especially in the cæcum. Twining of the small intestine around the colon, or some other portion of itself, sometimes occurs. We also find the intestines twisted through lacerations in the omentum, strangulated by pedunculated lipomata and other new growths on the mesentery, and compressed by overloading of neighbouring intestines. Constriction of intestinal loops occurs in the inguinal canal, in some old-

standing rent of the diaphragm, in the foramen of Winslow, in crevices of the omentum, the mesentery or of the broad uterine ligament, and between ribbon-like, old peritonitic adhesions. The intussusception most frequently met with is that of the small intestine into the cæcum. Rupture of the stomach generally occurs along the great curvature, at the spot where the serous covering is continued into the layers of the omentum, and may extend to three-fourths of its length. The edges of the rupture are inflamed, swollen and charged with blood, serrated and irregular. The rent in the serous covering is usually longer than that in the mucous membrane. Food is found lying between the several layers of the wall. The contents of the stomach are partly bloodstained, and penetrate into the abdominal cavity. Laceration of the intestine usually takes place in parts where fæcal stasis has occurred. Sometimes only the mucous membrane is torn, and the intestinal contents penetrate beneath it, so that it is raised from its support to a considerable extent; or the contents burst through the muscular wall on the side of the mesentery and penetrate between the mesenteric layers. Ruptures of the diaphragm show very ragged edges, between which a part of the stomach or bowels projects into the thoracic cavity.

The intestinal mucous membrane is sometimes hyperæmic, at other times inflamed, the inflammation being either of a catarrhal, croupy, diphtheritic or hæmorrhagic nature. Occasionally it is necrotic and ulcerated. The sub-serous and submucous connective tissue is infiltrated with blood, the contents are also bloody, the intestinal walls swollen, tender and friable. The peritoneum exhibits the utmost variety of inflammation from the sero-fibrinous to the malignant and ichorous. In the mesentery are numerous hæmorrhages. The mesenteric glands are swollen and charged with blood. The anterior mesenteric artery is frequently aneurismal, and contains the larvæ of *strongylus armatus*, while in the intestinal arteries are numerous thrombi and emboli. The general changes consist in hyperæmia of the abdominal organs and also of the lungs (the latter from overcharging), œdema of the lungs, hypostatic inflammation of the lungs, hæmorrhages of the pleuræ and meninges, parenchymatous degeneration of the heart, kidneys, liver and spleen, and, lastly, in the presence of dark, almost black, tar-like blood, rich in bacteria.

General Remarks on Causes of Death.—If, on dissec-

tion of a fatal colic case, a gastric or intestinal rupture, an extensive enteritis or peritonitis be found, there can then be no doubt as to the cause of death. Very often, however, a case of colic may end fatally within twelve to twenty-four hours, without any such changes having in that short time been produced in the abdominal cavity as would explain the sudden death. The following causes of death are all possible in such a case.

1. *Absorption of toxic material into the blood.*—This may either be of a chemical or septic nature, or a ptomaine or leucomaine poison. It has already been provéd by experiment that in the fluid contents of an imprisoned hernia, micro-organisms can be detected long before necrosis of the intestinal wall sets in. They arise from the interior of the bowel, for both bacilli and cocci penetrate the wall of the intestine and produce fatal peritoneal sepsis, and generally without any accompanying suppurative peritonitis. The liver appears in such cases to be the most important transitional depôt for the septic matter. This organ is found to be in an extreme state of septic corruption, sometimes even immediately after death, and before natural putrefaction could have commenced.

2. *Compression of the lungs, followed by œdema, with suffocation.*—Caused by pressure of the distended intestines upon the diaphragm. The proof of this lies in the frequent detection of œdema of the lungs and other symptoms of suffocation at *post-mortem* examinations.

3. *Blood poisoning by carbonic acid in tympanites.*—This occurs when the pressure (or tension) of the carbonic acid in the blood falls below that of the intestinal gases (which consist chiefly of the same acid); for then, in accordance with strictly physical laws, the blood absorbs carbonic acid.

4. *Increase of arterial blood pressure, with consecutive brain and lung apoplexy,* consequent upon the pressure of the distended bowels upon the abdominal aorta and larger veins.

In addition to these causes, which may all prove fatal, other explanations were formerly adduced. Thus heart shock from extreme pain was held to cause death, but this has little probability to commend it. Phenol poisoning of the body, through absorption of large quantities of phenol developed during abnormal bowel fermentation, was also thought to be a cause of death. But Tereg has proved that during colic the absorption of phenol is rather diminished than otherwise. The commonest causes of a fatal ending may be sought, therefore, in sepsis, œdema of the lungs and blood-poisoning by carbonic acid.

General Treatment.—The special treatment of colic due to specific causes will be dealt with later under separate headings, and it will therefore suffice to give here an outline of the general measures to be adopted in cases of colic. The prophylactic measures may be inferred from the nature of the causes. In order to guard against the colic which so frequently results from litter-eating in the Prussian army, hay and corn have been recently added to the rations, and molasses has been given with conspicuous success. The first provision requisite for a horse suffering from colic is a wide and spacious enclosure in which the animal can fling itself about without injury, and a plentiful supply of dry straw. Then the body of the animal should be rubbed all over with straw and woollen rags, in order to stimulate blood circulation and intestinal activity. This may be rendered more effective by sprinkling the skin with spirits of camphor, to which turpentine may be added. In slighter cases, when the colic symptoms are not too severe, and weather permits, one may try to stimulate the peristaltic action of the intestines by a little gentle exercise; this latter is specially advisable in cases of chronic constipation-colic, but all severe exertion, such as trotting or galloping, is to be avoided. In severe attacks of colic no attempts at movement should be made, as they only do harm.

In every case of colic the rectum should be thoroughly explored, not only as an aid to diagnosis, but in order that fæcal masses may be removed, the bowel, where displaced, put back into position, and the rectal muscles massaged. The omission of this highly important procedure, whether due to negligence, superficiality, prejudice, or a false sense of modesty, cannot be too severely censured. Clysters and water-injections (irrigations) should be employed with the object of softening the fæces and promoting peristalsis. Glycerine enemas, that is to say, the injection into the rectum of 30 to 75 grains of glycerine, have (like soap-clysters) only a local effect, and produce no stimulus of peristaltic action in the deeper intestines.

The practice among veterinary surgeons of introducing large quantities of water into the rectum is very old, but its effects have been much exaggerated. It has been asserted that ten gallons and more may be injected, and that the water will penetrate as far as the cæcum. But the fact is that any rinsing of the intestinal canal beyond the rectum itself can seldom be effected, and that more than two gallons can rarely be retained. Dreymann has recently introduced an irrigator, the tube of which is provided with a piston and two handles. By this means regurgitation is prevented, and it is possible to inject by slow degrees as much

as 40 to 80 pints of liquid. The temperature of the water thus used should, in acute and feverish colic, be *cold*, but in chronic cases *warm*. With this internal application of water, cold or warm fomentations of the abdomen should also be employed.

Moderate rolling about can certainly not harm a horse suffering from colic, and in cases of twisted intestines may even do good. But, on the other hand, violent rolling, impetuous throwing itself upon the ground, and, indeed, all excessive restlessness must be controlled as much as possible, as it may cause rupture of the tightly-packed stomach or intestines. The best sedative, and, therefore, also a specific remedy against spasmodic colic, is an injection of morphia (5 to 8 grains).

If the abdomen be greatly distended with gas, do not delay to puncture the intestine, an operation which probably never causes any injury, if performed with due antiseptic precautions. Blood-letting is also sometimes necessary if apoplexy of the lungs or brain be threatened. Internal administration of so-called gas-absorbing remedies (spirits of ammonia, lime-water, sulphuretted potash and hyperchloride of sodium) is but of doubtful effect.

For diminished peristaltic action and delayed evacuation, many remedies are employed. In general they are the same as have already been cited in connection with the constipation which accompanies intestinal catarrh. Sulphate of eserine (¾ to 1½ grains) given hypodermically in 1½ to 2½ fluid drachms of water, as recommended by Dieckerhoff, is deservedly highly spoken of. The use of eserine must not, however, be indiscriminate, for it is contra-indicated in cases of spasmodic colic, because of its tetanic and spasm-producing effect; also in cases of acute colic produced by over-feeding, in wind colic and in graver cases of colic arising from over-feeding, wind and constipation, on account of the danger of rupture of the stomach or intestines. It is stated that since the introduction of eserine the cases of rupture and volvulus of the small intestine have increased in number. It is therefore wiser to adhere to the smaller doses (¾ to 1 grain) rather than to the larger (1 to 1½ grains). As, moreover, a few instances of poisoning, as well as of abortion in pregnant mares have occurred (due probably to the inconstant nature of the drug), it is wise to use eserine with caution. With horses suffering from lung disease is this especially advisable, owing to the risk of œdema. A combination of eserine (1½ grains) with pilocarpine (3 grains) is suggested

by Ellenberger, and has been found efficacious. The object of the latter is to stimulate bowel secretions, and to liquefy the contents of the intestines. As a substitute for eserine, which is easily decomposed and very inconstant, and for pilocarpine, which is costly, Fröhner recommends hydrobromide of arecoline in subcutaneous doses of ¾ to 1½ grains. Dieckerhoff recommends barium chloride, but, owing to its unfavourable action upon the heart the doses should be regulated with great caution. It was formerly the custom to administer the drug in single doses, 7 to 20 grains when injected, and 90 to 150 grains when given by the mouth. It is now considered preferable, however, to give repeated small doses. Thus, 4 to 7 grains may be injected, the dose being repeated after a quarter of an hour, and so on. Opinions differ considerably as to the value of treatment with barium chloride; by some it is said to have a rapid laxative effect, by others it is credited with unreliability and toxic by-effects. In the Prussian army, the employment of barium chloride is either forbidden or permitted only in isolated cases.

Glauber's salt and tartar emetic have long been used, the former in 8 to 16 oz. doses, and the latter in 1 to 2 drams. They are frequently given in combination, the formula being somewhat as follows: Antim. pot. tart., 90 grs.; Soda sulf., ½ lb.; Pul. sem. fœnicul. et. rad. alth. q. s.f. electuar. The following are also in common use:—Aloes, 4 to 8 drams; calomel, 1 to 2½ drams; castor oil, ½ to 1 pint; linseed oil, 1 to 1½ pints; and in obstinate cases croton oil, 15 to 20 drops is used occasionally.

The above-mentioned drugs are not required in every case of colic. Indeed, it is a well-known fact that a moderately large percentage of colic patients recover without any medical treatment. It would, however, be a mistake to infer that in colic a waiting policy is always best.

We cannot endorse the uniform treatment of all colic cases with one and the same remedy, such as Lemke and Eckl recommend (viz., morphia), Adam (cold water douches), Luelfing (a mixture of aloes, Glauber's salt and seeds of the Trigonella Folium Græcum). Every case requires individual treatment.

The physical treatment of colic and allied conditions in horses.—Under this title Dr. Sobelsohn, of Vienna, has recently (1902) issued a very sound work, which forms a valuable addition to the therapeutics of colic. The advantages which he considers are to be gained by the employment of the physical method may be briefly summarised as follows:—

1. The repeated exploration of the rectum, which forms an essential feature of the mechanical treatment, facilitates diagnosis.

2. Should there be deviation, invagination, or incarceration of the

rectum (and only exploration will reveal their presence), the owner, having been made aware of the hopelessness of the condition, may be induced to consent to operative measures, the treatment thus passing into the domain of surgery.

3. The therapeutist is not only enabled to control the effects of his treatment, but is in a position to regulate and arrange it upon a strictly causal basis.

4. The method has no harmful influence, and may be employed without reference to the state of the heart and lungs.

5. A combined mechanical and irrigation treatment, applied directly to the bowel itself, possesses the following special advantages: (*a*) it removes the constipation; (*b*) it facilitates the expulsion of wind from the stomach and intestines; (*c*) in cases of embolism it provides, until circulation is restored, for the removal of fæcal masses and accumulations of gas, without provoking violent and dangerous peristalsis; (*d*) it permits of the emptying of the painfully-distended bladder more rapidly than by any other process; (*e*) it exercises a soothing and painless influence; and (*f*) it is the only method which offers a possibility of successful treatment under certain circumstances, of deviations and other conditions generally regarded as incurable.

I. COLIC DUE TO OVER-FEEDING.

Symptoms.—This form of colic is distinguished by violent and long continued paroxysms, and often by sudden death. The breathing is generally very heavy, the abdomen greatly distended, abnormal positions are common, especially that assumed by dogs when sitting, by which the animals undoubtedly seek to relieve both diaphragm and lungs from the pressure of an over-loaded stomach. Characteristic symptoms, though not very frequently noted, are—belching, retching, straining and vomiting. These sometimes end in rupture of stomach or diaphragm, which is recognised at once by the sudden cessation of the colic symptoms, and general signs of collapse.

Diagnosis.—Here, strict attention must first be given to the history of the case, and to verification of the fact that the colic arises from too liberal feeding. Without this knowledge it might be difficult, or even impossible, to distinguish it from any other form of colic. For even the characteristic sign of overfeeding, viz., vomiting, may arise from other pathological conditions of the stomach or intestines; the same applies to rupture of the stomach or diaphragm.

Prognosis.—This must generally be made with caution, the more so, as the horse is unable, or only rarely able to relieve

its stomach, as other animals do, by vomiting, and is liable to rupture either stomach or diaphragm if it throws itself down with violence.

Therapeutics.—The treatment of colic, arising from over-feeding, consists first of all in emptying the rectum by manual exploration, and in frequent administration of clysters, so as indirectly to expedite the advance of the contents of the stomach. In slight cases, eserine and arecoline may be given in small doses (¾ gr.), as well as tartar emetic (½-4 drs.) to stimulate peristaltic action. In all severe cases, eserine and arecoline must be avoided, owing to the great danger of stomachic rupture. In such instances, we must give morphia injections (7 grs.), to prevent the animal from plunging or flinging itself on the ground. Dieckerhoff recommends barium chloride (7–20 grs. if injected into the veins; 1½–2½ drs. if in draught). Marck has latterly employed a stomach-sound in the treatment of overloading of the stomach and tympanites in horses. The animal is kept in a standing position, with bent head, raised chin and tongue drawn out; the mouth-piece is put into place and a six-foot probang is then introduced. After the introduction of the tube into the gullet, nearly all horses roar.

Vomiting.—Special interest pertains to the symptom of vomiting, which occasionally occurs in cases of colic caused by over-feeding. The act of vomition—which, as is elsewhere explained, is peculiarly difficult for the horse—happens sometimes in these cases, owing to the abnormally distended condition of the stomach and the relaxation of the otherwise firmly closed cardia. The phenomena which have thereby been noticed are as follows: After spasmodic contraction of the belly and neck muscles, accompanied by an in-drawing of the head to the breast and lowering of the neck, a fluid is discharged from the mouth and nose, of varied consistency, according to the food consumed, and generally greenish-yellow, frothy and sour in smell. The quantity is very irregular. Sometimes a whole stable-bucketful is discharged at once, and at other times, after hours of vomiting, very little fluid is emitted. During this vomiting the animal sweats, puts its feet under the body and shows widely-opened, staring eyes. When the fit is over, it seems much exhausted, sways about and trembles and coughs for a long time. Sometimes it does not reach the point of vomiting but only slavers, at the same time straining, retching and belching. With regard to the prognostic signification of vomiting, it is a mistake to view it as an unfavourable, or indeed ominous symptom. In many cases it undoubtedly relieves the stomach, and in very many others no injurious after-effects have been noticed. Nevertheless, it must in general be considered as a sign calling for great caution, as, with an abnormally distended stomach, rupture of that organ may easily take

place; and also because other serious affections of the gastro-intestinal canal may be the primary cause of vomiting, such, for instance, as an ulcerated stomach, stenosis of the intestine or peritonitis. Vomiting also occurs in certain diseases of the œsophagus, especially distension as well as in inflammation of the diaphragm. Finally, vomiting also follows sometimes on the administration of eserine, arecoline, pilocarpine, morphium, chloroform, chloral hydrate and barium chloride. In some cases it occurs without apparent cause.

Rupture of the Stomach.—This happens during the course of colic produced by overfeeding, owing to the excessive distension of the stomach and final rending of its walls; or to violent shock caused by the animal flinging itself down; or, finally, to spasmodic contraction of the abdominal walls during vomiting. It happens also when vomiting is prevented by the firmly closed cardia, whereby "auto-rupture" of the stomach is produced by its own excessive muscular contractions. On the mutual relation of vomiting and rupture of the stomach erroneous opinions were long current, for people believed that the former was the result and not the cause of such ruptures. But it is quite impossible that after the stomach has been ruptured, and its contents emptied into the abdominal cavity, any portion of those contents should find their way out through the mouth. When, therefore, vomiting and stomach rupture are found together, the latter can only be regarded as a consequence of the former. On the contrary, though, where there is only a partial rupture of the walls of the stomach, as, for instance, when only the muscular coat, or a portion of the same, is rent, this rupture may itself be a cause of vomiting, if it coincide with a paralysis of the cardia. The phenomena of bowel and stomach rupture are difficult to differentiate. Sometimes they are preceded by vomiting, retching and eructation. Generally, however, rupture shows itself suddenly in a complete collapse at the height of a violent colic attack. The signs of colic disappear, the abdominal sounds become inaudible, the pulse is feeble and extremely rapid, the extremities grow icy cold, the body is bathed in cold sweat, and the internal temperature sinks below normal or rises rapidly. The animal sways and trembles, sometimes neighs, assumes extraordinary positions, appears as though stupefied, draws its feet under the body, experiences great difficulty of breathing and shows an anxious and staring expression and a stiff gait. Sometimes a viscid, greenish-yellow mucus exudes from the mouth. Death generally occurs in a few hours, but sometimes not for several days, if the rupture be only partial, or if some organ, such as the omentum, lie across the rent. Such cases, however, are rare.

Rupture of the Diaphragm.—This arises from the pressure of an over-filled stomach, or from the animal suddenly flinging himself on the ground, and is facilitated by an antecedent hernia or inflammation of the diaphragm. In many cases rupture of the diaphragm occurs without any previous symptom of colic, as after a fall, over-exertion when at work, or from unknown causes. On dissection one finds that either the stomach, small intestine, colon or omentum, is protruded through the opening into the thoracic cavity, and in chronic cases is sometimes found grown fast to the lungs, ribs or pericardium. In such cases the inserted portion of the bowels often becomes constricted. The

symptoms of rupture of the diaphragm are, first of all, excessive difficulty of breathing, which may amount to suffocation, and in which the abdominal muscles are spasmodically contracted. The animal then sometimes has violent fits of colic, strikes extraordinary attitudes, especially that of sitting like a dog, sways about, sweats profusely, coughs, breathes with a snore, is unable to neigh, but can only emit feeble noises. If a bowel has slipped forward into the thoracic cavity, one can hear on percussion of the thorax, frequently as far as the shoulder, a clear, drum-like sound, and on applying the stethoscope, a series of gurgling, rumbling and splashing bowel-sounds ; the usual vesicular breathing sounds meanwhile being inaudible. These latter symptoms render the diagnosis certain under all circumstances. Nevertheless, in rupture of the diaphragm they may be absent, as no portion of the bowels, or only very little, may have fallen forwards, occupying only the lower part of the cavity. Moreover, one must be careful not to confound any unusually loud peristaltic murmur of normally situated intestines with the above noises. The progress of a rupture of the diaphragm is generally very acute, and death from suffocation often happens in a few minutes, or within half an hour. In rarer cases it is chronic ; the animal apparently recovers, sometimes quickly, sometimes after a colic lasting for several weeks. But it relapses later, often at quite regular intervals, say, for example, from four to fourteen days, or after the slightest exertion. Others are greatly distressed, particularly in going downhill, and others never lie down again. Finally, permanent difficulty of breathing and gradual emaciation may result.

II. COLIC FROM EMBOLISM AND THROMBOSIS IN HORSES.

Historical Notes.—Aneurism of the anterior mesenteric artery, caused by worms (worm-aneurism), was very early associated with colic by medical men. Thus, Rigot (1829), and Schutt (1835) discovered aneurism after previous colic. Bruckmüller speaks of cases "in which aneurism of the mesenteric arteries is said to have produced the colic so common in horses," and is of opinion that a causal relationship between them cannot be denied. Prehr seeks in one case the origin of the colic in the actual obliteration of the anterior mesenteric artery. Bouley says that bloody excrements and intermittent colic are special elements in the diagnosis of aneurism of the mesenteric arteries. Köhne believes that in a colic case watched by him, and caused by stoppage of the anterior mesenteric artery, the large intestines, and especially the blind-gut, were reduced to a state of paralysis. Hahn assumes as the cause of a periodically returning colic, the aneurism of the anterior mesenteric artery revealed by dissection. Finally, Cornevin described

a very fine case of thrombotic colic, with occlusion of the arteries of the ileum and cæcum.

From these particulars it is plain that the relations of cause and effect between aneurism and colic had been sought, and at least partly found, prior to the publication of Bollinger's work, "Colic in Horses, and Worm-Aneurism of the Intestinal Arteries." Notwithstanding, great merit is due to Bollinger for having clearly established the relationship by his extensive researches.

Anatomical Notes on Worm-Aneurism of the Anterior Mesenteric Artery.—Hering showed in 1830 that this aneurism frequently occurred in the horse. Indeed, he maintained that a horse is more likely to have an aneurism than not. According to the calculations of Bruckmüller, Roell, Bollinger and others, 90-94 per cent. of all horses suffer from the aneurism in question. It seems to be most common in aged animals. Thus, Ellenberger only found a single horse free from aneurism among 85 used for dissection. It is met with in foals of three months, and recent experiments show that it may be developed during fœcal life (Poppel). The nematoid larva passes from the bowel of the mother by way of the mesenteric artery into the blood-stream, is carried into the umbilical artery, and is thence introduced into the circulatory system of the fœtus.

Aneurism of the anterior mesenteric artery, which supplies with blood all the small and large intestines, except the middle and hinder regions of the rectum, is by far the most frequent. It is rarer to find the posterior mesenteric, or the aorta affected. The walls of the aneurisms are mostly very much thickened, especially the middle coat and the peritoneal covering. In form they are usually evenly expanded to a long oval, and bottle or spindle-shaped, more rarely bag-shaped, as large as a man's head, and even as much as a foot in length. The inner coat displays the most varied inflammatory and degenerative changes, such as swelling, fatty degeneration, calcification, ossification, ulceration, and sclerosis of the connective tissue. The contents consist mainly of a parietal, stratified, and often vascular thrombus, which may entirely obstruct the lumen of the aneurism, and even project itself into the various branches of the several arteries, and sometimes even into the lumen of the aorta. Not unfrequently it is very loose and friable, and at times even softened and degenerated. In addition to the

thrombus a few larvæ of the *strongylus armatus Rud.* are found; on an average nine in each aneurism. The presence of these worms is the proximate cause of the chronic changes in the inner coat, and consequently of the thrombus and aneurism also. Their number may be very considerable (100-200 and more). As to the manner and period of their immigration nothing certain is known. [For further particulars of *strongylus armatus* see chapter on " Intestinal Worms."]

Pathogenesis of Worm-Aneurism.—It has been generally held up to the present that aneurysma verminosum is caused by the continued irritation by worms of the tunica intima. But Sticker has recently shown that it is to changes in the media that aneurisms are due. His investigations show that the larvæ penetrate by way of the vasa vasorum into the muscular structure of the arterial wall, giving rise to hæmorrhagic embolic infarct, acute mesoarteritis, periarteritis fibroplastica, acute endocarditis and slight thrombosis. In the course of the progress and further development of the worms, larger quantities of intimal and medial tissue are lost, causing pronounced thrombosis and the distension and chronic degeneration of all three epithelial layers.

Sticker has also shown that the young worm which appears after the sloughing of the larva found in aneurism of the anterior mesenteric artery, is the earlier form of the smallest of the three varieties of palisade worms which occur in the large intestine of the horse. This variety is known as *sclerostomum bidentatum* (*aneurysmaticum*), on account of the two hook-shaped attachments (teeth) with which the head is furnished. It is also called the " vein-worm " (Aderwurm).

Worm-Aneurism in its Relation to Colic.—Apart from the fact that aneurism of the anterior mesenteric artery hinders the due nourishment of the bowels by checking the supply of blood, and thereby pre-disposes to colic, the bowel is further permanently exposed to a three-fold danger. First of all, the thrombus may entirely choke up the lumen of the anterior mesenteric artery (this is rare); or it may prolong itself into any branch of a bowel-artery; or, finally, a severed particle of the thrombus may be carried along by the flow of blood, as an embolus, into one of the intestinal arteries. In all three cases, unless sufficient collateral circulation be developed,

changes are set up, as has been abundantly demonstrated by experiment. First, then, an arterial anæmia is produced in the isolated vascular region, which is followed, owing to the interrupted arterial blood-pressure, by a backward venous flow of blood, accompanied by serous and sanguineous infiltration into the walls and lumen of the intestine, which directly produces paralysis of the bowel. This paralysis, in its turn, causes stagnation of the intestinal contents, with abnormal fermentation, and excessive production of gas. It may also occasion volvulus and invagination of the bowel, as the healthy and active intestines twist themselves about the paralysed and passive portion, or project themselves into it. As volvulus of the left part of the double colon is so frequently a result of thrombosis and embolism, and as this can only be presumed to arise from active movement of this part, which must be first caused by disturbances of the circulation, we are forced to the conclusion that arterial anæmia causes at first a stimulated movement, which sets up this displacement. We do not think that in this case the volvulus of the left portion can be explained by the intensified activity of the neighbouring healthy intestinal parts.

Finally, paralysis of the bowel leads to inflammation of the same, and to rupture, both of bowels, stomach and diaphragm, as the anti-peristaltic intestinal movement, set up in the disabled part, fills the stomach to bursting point with food matter, and the bowels, being distended with gas, either split themselves, or by their pressure cause rupture of the diaphragm.

Anatomical Relations of the Anterior Mesenteric Artery.—These are very important for the due understanding of the origin of thrombo-embolic colic. As is known, this artery springs as a short strong stem, about 1½ inches long, from the posterior aorta. Immediately above its source the anterior mesenteric artery throws off—1st, the superior colic artery, in a forward direction ; 2nd, backwards and directly opposite the exit of the foregoing, the 17 to 20 small intestinal arteries. From this point onwards the somewhat narrowed trunk of the anterior mesenteric artery bears the name of the ileo-cœcal artery. This trunk is usually the seat of worm-aneurism. It divides next into four branches—the front one, which is strongest and forms to some extent the continuation of the artery itself, is the inferior colic artery. Then follow one after the other the upper and lower cœcal arteries, and finally the artery to the ileum and cæcum.

As a rule, the ileo-cœcal artery is not completely blocked by the aneurismal thrombus, so that the terminal arterial branches continue to be fully supplied with blood, and no severe colic can ensue. Only when the ileo-cœcal artery is entirely closed (which rarely happens) is the

cæcum deprived of blood. In the case of the colon, however, on which only the inferior colic artery is blocked, the upper one, which anastomoses with the lower, remains free, and by itself continues to supply the colon with blood. The occlusion, therefore, of one colic artery does not of itself suffice to produce a fatal thrombo-embolic colic. (This is important in forensic cases.) *Both* colic arteries are only then affected when the aneurism implicates the source of the anterior mesenteric artery, which is rarely the case. Should this happen, then embolism of the arteries of the small intestine may follow. These, however, generally escape. On the other hand, transient colic may arise from sudden stoppage of *one* colic or cœcal artery.

If portions of aneurismal thrombus become detached, emboli may occur, but generally only in the terminal arterial branches, especially in the inferior colic artery. This one-sided embolus does not produce fatal colic, because the upper colic artery remains patent. Neither does embolus of *one* of the two arteries of the cæcum end fatally. For they, too, are connected, and the gut is moreover nourished by a small vessel which reaches it from the inferior colic artery. The occlusion of one cœcal artery is not of great importance, as long as the obstructing clot is a small one. The condition becomes dangerous when a large portion of the artery gets blocked or when a number of clots become piled one above the other a certain distance apart. Pronounced embolism of this description, of even one of the cœcal arteries, prevents the flow of blood to the neighbouring blood-vessels in a quantity sufficient to supply the walls of the intestine at that particular spot with sufficient nourishment for working purposes. In consequence, the contents of the bowel become massed at this point, while the intestinal wall shows œdematous swelling, due to the circulatory disturbance.

The arteries of the small intestine finally separate near the bowel into two curved branches, each of which flows into the neighbouring anterior or posterior branch. From these curves straight channels run to the bowel. If, therefore, *one* embolus fix itself above the bifurcation of an arterial branch, the flow of blood is not thereby prevented, because the vascular curves and the branches springing therefrom are mutually connected.

Symptoms and Course.—The course of thrombo-embolic colic may vary greatly.

1. *Acute Colic Ending in Recovery.*—This not uncommon form is characterised by frequent and recurrent attacks, without any apparent cause, which bear much resemblance to rheumatic colic, being sometimes extremely painful, but generally brief. Probably they arise from partial embolisms of the intestinal vessels (colic arteries), which are gradually worked off by anastomosis. As positive diagnosis is naturally difficult, they are often mistaken for other forms of colic.

2. *Acute Course with Unfavourable Ending.*—This refers to all cases in which thrombosis or embolism produces displacement of the intestine. It is here also characteristic that the

manifestations occur without any outwardly discoverable cause. In contrast to other kinds of colic, the pains are often quite trivial. This is perhaps to be explained by paralysis of the local nerves by complete stoppage of circulation. In other points the symptoms agree with those of constipation colic caused by internal displacements, which are neither characteristic nor constant. Death follows either from rupture of stomach or bowels, or else from sepsis.

3. *Chronic Course with Relatively Favourable Ending. Chronic Embolic Bowel-Catarrh.*—Consequent upon occlusion of intestinal arteries, hyperæmia, due to congestion, is first developed. Should this, owing to its situation, not lead to serious changes, the hyperæmia may last a long time, and lead eventually to chronic intestinal catarrh, as, for instance, has been noticed with congestion in the region of the mesenteric vein (portal vein) after liver disease. (" Embolic intestinal catarrh.") The course of the complaint is then generally as follows:—After relief of the first attack, the increase of temperature and pulsation disappears, but the peristaltic action continues to be retarded, evacuation is suppressed, less food consumed, and in general the condition is unsatisfactory. After one or two days low fever reappears, with intermittent, though slight, symptoms of colic, which, however, may be totally absent. In this long-drawn-out fashion it may continue for several days and even weeks (chronic, creeping colic) ; in one case it lasted twenty-two days. Occasionally this chronic intestinal catarrh passes into chronic emaciation and cachexia. It ends more frequently in death with the appearance of sepsis.

4. *Chronic Course with Unfavourable Ending. Development of Thrombo-Embolic, Hæmorrhagic Inflammation and Necrosis of the Intestinal Mucous Membrane. Hæmorrhagic Infarction of the Bowels.*—The pathogenesis of this hæmorrhagic inflammation may be explained by thrombo-embolic occlusion of small intestinal arteries, in conjunction with a local worm-aneurism, whereby only smaller portions of the mucous membrane of the intestinal canal suffer from the interrupted circulation mentioned before (arterial anæmia, vascular engorgement, hæmorrhagic congestion, necrosis). The chief symptoms are a greatly diminished appetite, which indeed may be altogether lost for days, increased thirst and retarded evacuation, with distinctly audible peristaltis. The fæces are at first small pellets; later slimy, fœtid and pulpy, and often bloody (blood-colic) ; the urine is very acid and contains phosphates

and albumen; intense and lasting fever (105° F. and more) shows itself; the pulse grows weak, but very rapid (60–70, and later as high as 90–100 per minute). The weakness increases, accompanied by wasting, a falling in of the hind-quarters, alternate dung-balls and pulpy fæces, discoloured urine and affection of the sensorium. Sometimes colic recurs immediately after eating, and the condition grows worse. Occasionally towards the fatal end remarkable paroxysms of fever are noted, viz.: violent and general quivering of the muscles and shivering; the extremities become icy cold, and the mucous membranes, where visible, turn pale; the breathing is difficult, rapid and rattling; palpitation of the heart sets in, and the internal temperature rises. The duration is generally lengthy. Often it is preceded by a prolonged chronic colic of the intestines. The actual hæmorrhagic inflammation may last for days and even weeks. Death finally results from absorption of septic matter from the bowel into the blood-vessels; from peritonitis or chronic marasmus. Recovery is rare, but proceeds from detachment of the necrotic scab and healing of the lesion; though chronic ulcers may remain behind as a consequence. In any case the stage of convalescence is very prolonged.

Rupture of Worm-Aneurism.—In a foal of eight months which died suddenly, never having shown symptoms of colic, Francke found an aneurism as large as a man's head, containing 200–300 larvæ. It had grown to the right upper colon, and was ruptured in such a way as to cause hæmorrhage into the bowel. Labat and Cadéac observed a similar occurrence in the case of a mare.

Anatomical Notes.—Dissection reveals very characteristic changes in hæmorrhagic congestion of the bowels. Besides the usual signs of sepsis in the stomach, with swelling, injection and ecchymosis of the mucous membrane, one finds small, reddish-black blood coagula attached to plugs (thrombi) of small eroded vessels of the mucous membrane and submucosa; in the small intestine is a thin, blood-coloured fluid, with dull brown-red infiltration of the mucous membrane; the same also in the large intestine, whose walls in circumscribed places (sometimes at its junction with the mesentery) are swollen by a serous-hæmorrhagic infiltration to thrice their normal thickness, or even more. Very seldom are embolic changes found in the rectum. At the spots where this hæmorrhagic congestion is greatest the mucous membrane is friable

and covered with a necrotic scab, strongly infiltrated with blood round its edge. At the base of this scab one can very often find, on more careful investigation, a thrombotic intestinal artery and follow it up centrally, whereby the existence of a worm-aneurism of the anterior mesenteric artery may be demonstrated. In addition gelatinous infiltrations may be found in various parts of the abdominal cavity (especially the anterior root of the mesentery), and large quantities of bloody serous or ichorous fluid in the peritoneal cavity.

Treatment.—The treatment of thrombo-embolic colic is symptomatic. It consists in attempting to remove the morbid conditions arising from the intestinal paralysis, as the locality of the vascular changes in question is not accessible. As these consist chiefly in an accumulation of dung, which, if too long neglected, produces local inflammation and necrosis of the mucous membrane, it is first of all needful to remove this coprostasis by the use of purgatives, such as calomel or neutral salts. Whether physostigmine be also applicable is doubtful, owing to the fact that the intense bowel-contraction which it produces may easily cause the affected parts of the bowels to be twisted or displaced by the unaffected intestines. Diligent massage is also advisable, and gentle exercise of the sick animal, so as to remedy any stoppage of circulation, together with constant cleansing of the rectum by hand and by injection of water. The treatment of thrombo-embolic, hæmorrhagic *gastro-enteritis* is identical with that of inflammation of the stomach (which see). The *dietetic supervision* of the sick animal, especially when convalescent, is of great importance. To prevent necrotic lesions of the mucous membrane calomel may be used, and various antipyretics are useful against septic fever. But, on the other hand, all drastic purgatives, such as tartar emetic, aloes or croton oil, must be avoided.

The Bollinger theory of the relation between colic and worm-aneurism of the anterior mesenteric artery has met with enthusiastic support from some and emphatic contradiction from others. Especially has Gerlach, in his "Forensic Veterinary Science," 1872, p. 531, declared himself strongly against there being any connection between the two. Röll ("Textbook of Pathology and Therapeutics") would admit within certain limits the extension postulated by Bollinger of thrombo-embolic colic to all sudden cases of colic which cannot be traced to distinctly provable causes. We ourselves are of opinion that the existence of thrombo-embolic colic has been clearly shown, and that it is by no means rare. But we hold that Bollinger certainly went too far when he declared that

50 per cent. of all fatal cases of colic are thrombo-embolic, and that every sudden case not traceable to any clear cause may be so classified.

III. CONSTIPATION-COLIC IN HORSES.

SUB-VARIETIES.—As many causes tend to produce constipation, so there are varied forms of this disease to be differentiated, which, however, cannot be clinically separated. These varieties of constipation-colic, which are only etiologically different, may be caused by :—

1. Accumulation of food in the bowels.
2. Gastric and intestinal calculi (stone-colic).
3. Displacement of the intestines.
4. Invagination of the bowels.
5. New growths producing stenosis.
6. Stricture of the bowels.
7. Dilatation and paralysis of the bowels.

I. CONSTIPATION-COLIC CAUSED BY ACCUMULATION OF FOOD IN THE BOWELS.

Etiology.—The causes of this complaint have been already cited in our general remarks. They are generally to be traced to some unusual form of food. Especially dangerous are an excess of bran, straw cut too short, or an overdose of straw (straw-colic), and particularly pea and rye straw if chopped too fine, tough-fibred clover, spelt-husks, beech-nut shells, bean-stalks, in a word, any indigestible cellulose horse-feed; furthermore, all kinds of relaxing food, and a too sudden change from pasturage to stable food, or exclusive feeding with maize. Insufficient exercise combined with full feeding, as well as defective mastication arising from defects of the teeth, are both causes of feculence. Foals sometimes during the first few days after birth show signs of colic arising from an accretion of meconium in the rectum. There are also numerous cases of constipation-colic caused by the combined exhibition of morphia and atropine in paralysis of the shoulder (inhibition of peristalsis and of the glandular secretions).

Anatomical Conditions.—The local changes produced by the accumulated fæcal matter in the obstructed gut are very diverse. The substances which have lodged there dry up more and more by absorption of their moisture into the bowels, and thus become sources of irritation to the surrounding membrane. This causes, in the first place, catarrhal inflammation of the mucous membrane, with profuse ecchymosis (hæmorrhoidal

catarrh), and, later, diphtheritic and necrotic conditions. According to Ernst, the diphtheritic inflammation of the mucous membrane begins with small spots, at first grey and afterwards greyish yellow, or even white, which show especially on the projecting portions, and make its surface look as though sprinkled with bran. These spots are caused by a superficial necrosis of the membrane, which leads to the formation of ulcers and pustules ; only in rare cases is there any deeper necrotic injury of the tissue resulting in perforation. A further consequence of fæcal stasis is rupture of the stomach and bowels. Thus fæcal stasis in the cæcum (unless removed) appears regularly to produce rupture of the affected bowel, and frequently in the colon and rectum. Fæcal stasis in the passage from the ileum to the cæcum is, in a moiety of cases, followed by rupture of the stomach, in which a so-called anti-peristaltic intestinal movement, arising in one of the obstructed parts, drives the contents of the bowel back upon the stomach, overfills it, and causes it finally to burst, unless it be relieved by vomiting. Food accumulations occur most frequently in the ileum, just before its point of discharge into the cæcum.

Symptoms.—The manifestations of colic produced by accumulation are less marked than in other forms. The pains are usually mild, the animals generally lie still, and groan occasionally. They cease to take food. The passage of fæces is delayed, the peristaltic movement is suppressed, the separate dung-balls are very dry, small and often covered with slimy shreds. On examining the rectum, during which the animal generally presses and strains violently, the gut will be found full of solid fæces. As the overcharged gut presses upon the bladder, the animal makes frequent attempts to urinate, whereby its abdomen becomes gradually fuller of gas and is at last distended. The disease runs sometimes for a considerable time as a simple constipation without any colic symptoms (as with dogs) ; but if it last very long, the latter signs begin to show themselves. The complaint is always of prolonged duration. Usually it lasts several days, very frequently two or three weeks, and in some cases six or seven weeks ("chronic" colic). Meanwhile there are no true remissions, such as are characteristic of thrombo-embolic colic. Vomiting is rare. The symptoms of bowel rupture, which is too often fatal, closely resemble those described under the head of stomach rupture. In one case of very pronounced

tympanites, rupture of the stomach was complicated by rupture of the parietal peritoneum and general emphysema (Fiebinger).

Therapeutics.—The treatment consists in the administration of laxatives, also in massage of the abdomen, cold bandages, liberal injections of water, moderate exercise and low diet. Dose at first with Glauber salt, or artificial Carlsbad salts in ½ to 1 lb. doses. By some the use of castor-oil in combination with other oils is much recommended. They give for several consecutive days a mixture of 1½ pints of poppy-seed oil, or olive-oil, with 8–16 oz. of castor-oil. Also eserine (¾–1½ grs.), in conjunction with pilocarpine (4 grs.). Arecoline (1 gr.), as well as aloes (8–12 dr.), are often used with good results. Eserine seems, however, to be more suitable for milder cases of the complaint; in more serious ones it should not be used at all, or only with great caution, owing to the risk of bowel-rupture. Against tympanites, which sometimes ensues during constipation-colic, Albrecht recommends creolin (every three hours in doses of 1 dram, combined with laxatives).

Meconium colic.—Colic in calves, due to the retention of the meconium, generally makes its appearance on the second day after birth. According to Töpper, it is only to be treated by the mechanical removal of the meconium. He recommends for this purpose Masch's "Meconotorium," a spoon- or loop-shaped instrument. After the removal of the obstruction he gives 15½ grs. calomel in combination with 1½ oz. castor oil. Mensik's "rectal cleanser" serves the same purpose.

2.—CONSTIPATION-COLIC, FOLLOWING UPON GASTRIC AND INTESTINAL CALCULI (*stone-colic*) *and other foreign substances in the intestinal canal* (*sand-colic*).

Occurrence.—Stone-colic is, as a rule, rare. Among 1,882 fatal cases of colic in the Prussian Army (1889–93) only 25, or 1·5 per cent., were authenticated as stone-colic. It occurs most frequently among the horses of millers and bakers, which are almost exclusively fed upon bran and flour refuse. Bran, for instance, contains 1 to 2½ per cent. of phosphate of magnesia, which is the chief ingredient of these calculi. The small proportion of millstone dust, which is mixed with the bran, seems to be of less account. The so-called sand-colic must also be mentioned here. It arises from swallowing sand or minute stones, when drinking from shallow brooks or pools; from eating muddy or sandy fodder, on the way to pasture, or

after the stable gangway has been sanded. Finally, the animal's habit of licking itself when shedding its coat tends to the formation of balls of hair and consequent stoppage. Other foreign bodies, such as bits of cloth, sticks, buttons, screws, pieces of wire, needles, etc., when swallowed, are often causes of constipation.

Intestinal calculi, or enteroliths, consist for the most part of ammonio-phosphate of magnesia (90 per cent., Fürstenberg), also of phosphate and sulphate of calcium, phosphate of magnesia, silicates, chlorine compounds, traces of iron, as well as of organic substances (mucous, epithelium and particles of fodder). Their formation is explained by the combination of the phosphate of magnesia, so largely present in bran, with ammonia compounds in the intestines, which reach them from the ammonia-laden air of the stable through the drinking water consumed, and afterwards combine to form an insoluble compound. The core of the stone is usually some foreign substance, a grain of sand, or of oats, or the head of a nail, around which the accretion of phosphate is formed. So-called stomach-calculi are nothing else but bowel-calculi, which, by the anti-peristaltic movement of the bowels, have been forced back into the stomach ; for food remains so short a time in the latter that no calculus can be formed there in presence of its strongly acid contents. The formation of calculi usually occurs in the large intestine, and particularly in the greater curvature of the colon—much more rarely in the cæcum. Colin found, for instance, in 900 dissections, that the stone was in the colon in 23 cases, and only once in the cæcum. In addition to calculi, other accretions may lead to constipation, such as collections of sand or mud mixed with vegetable fibre, mucus, hair, etc., and there are so-called false calculi, which are such accretions coated over with phosphate. The local changes produced by bowel-stones are the same as those described in connection with fæcal stasis. In sand-colic large quantities of sand are often found in the intestines—30 to 50 lbs., and even more.

Symptoms.—So long as the calculi are free to move about in the bowel, they seldom give rise to symptoms of colic. Occasionally they are found unexpectedly in the *post-mortem* examination of horses who have died from other causes. In a case observed by Zschokke, that of an eighteen-year old miller's horse, which had never shown symptoms of either colic or any other affection, 46 lbs. weight of calculi

were found in the colon, one of the stones being thirty inches in circumference and weighing 22 lbs. Colic only arises in such cases when the stones are wedged in the rectum, or when they have become lodged in the colon, giving rise to diphtheritic inflammation of the mucous membrane. The symptoms of colic caused by calculi are frequently indistinguishable from those due to impaction. In some cases the attack has been preceded by several others, and these are often periodical in their occurrence. Individual attacks sometimes show a varying intensity as the stone (or stones) are worked along the bowel. An attack of colic is occasionally followed by a lingering intestinal catarrh. In general, the attacks are more severe in cases of stone-colic than of stoppage-colic, and the constipation is more obstinate. Vomiting and rupture of the stomach and bowels are more common in stone-colic. Its course is generally shorter, though it may at times be prolonged. The prognosis is much more unfavourable.

A positive diagnosis of stone or sand-colic can only be made when bowel-stones or sand are evacuated through the rectum, or after examination with the hand. Only with millers' horses, particularly if frequently affected, may the presumption be ventured upon that they are suffering from stone-colic, though other forms of colic, especially that which proceeds from overfeeding, are not uncommon with them.

Therapeutics.—Wherever it is possible to locate the concretions in the rectum, they should be removed with the hand. The treatment is the same as for colic caused by accumulation. Eserine must, however, in all cases of stone-colic be avoided, owing to the risk of intestinal rupture, but in sand-colic it may, on the contrary, be with advantage administered in small doses. In desperate cases, the firmly wedged stone may be removed by operation, if it can be located. Thus Félizet removed a stone the size of a child's head by flank incision. On the other hand, horses operated on by Hall, Hobday, Rickards and Dollar died after 24 to 27 hours. The proposal of Trasbôt to drive back the stone by generating carbonic-acid gas in the rectum by means of soda and tartaric acid could scarcely be carried into practice. French veterinary surgeons recommend for sand-colic in military horses the drinking of immense quantities of water—say 20 to 25 gallons daily, or in 8 days, about 160 to 200 gallons.

3.—CONSTIPATION-COLIC ARISING FROM DISPLACEMENT OF THE INTESTINES.

Anatomical Conditions.—The internal changes of position of the intestine consists of volvulus and twisting or invagination of the intestinal parts. Volvulus most frequently affects the colon (more particularly its left layers) and the small intestine. Volvulus of the cæcum is rather a kinking, with displacement of the point downwards and backwards, and a shifting of the base upwards and forwards. Volvulus of the rectum is rarer. Twisting or knotting arises from the coiling of the small intestine in loops around some portion of itself, or of the colon and rectum, whereby they are twisted round the mesentery. Or it is caused by omental cords wrapping around loops of the small intestine, and less frequently by the cæcum passing into the colon. Invaginations, finally, occur specially in congenital or acquired openings in the abdominal cavity, thus in the inguinal ring, in Winslow's foramen, in old fissures of the diaphragm, in apertures of the omentum and mesentery, in clefts formed by ribbon-like adhesions, the detached ureter, or adhesions of the omentum to the scar left by castration.

Etiology.—The real causes of these internal displacements are mostly obscure. Some certainly belong to the sphere of thrombo-embolic colic, and may be explained as already stated. Others arise from an unequal filling of certain bowel-loops with gas or with very heavy food, whereby these loops are constrained by gravitation to seek a change of position. Moreover, acute peristalsis, such as occurs in severe colic, appears often in the later stages of the colic to produce displacement. It is therefore not impossible, as many observers have supposed, that the use of eserine frequently produces displacement. Finally, the contortions and violent rolling of the animal are, in rare cases, a cause, although this origin is much too frequently assumed. The drinking of cold water, once so largely blamed, can scarcely be seriously considered as a factor.

Symptoms.—The phenomena have nothing characteristic about them. Persistent vomiting, lasting several days, is a symptom which may, however, be absent. The strange attitudes and signs of pain, movement to and fro of the head, lying on the back, the appearance of stomach or bowel rupture and absence of fæces, all occur in other forms of colic arising

from constipation. The duration varies greatly, according to the completeness or otherwise of the bowel stoppage. In the latter case it may last eight days, but in complete stoppages death usually occurs within a day. Often the colic signs are of a transient character, and return periodically, when, for instance, a bowel-loop is alternately temporarily imprisoned and released. Possibly the brief attacks of colic of so-called "night kickers" may be so explained. Occasionally violent rolling is followed by a sudden cessation of the symptoms.

A diagnosis of constipation-colic produced by internal displacement is possible only in rare instances, as when a strangulated inguinal hernia, or some other strangulation or twist, can be demonstrated from the rectum. Torsion of the colon and cæcum can sometimes be verified in this way. In rupture of the diaphragm the peristaltic murmur is heard very far forward upon auscultation of the thorax and this sign, together with an empty percussion-sound and the presence of extreme dyspnœa, is of assistance in the establishment of diagnosis. Finally, the fruitlessness of the treatment adopted may also guide in forming a diagnosis.

Therapeutics.—As a rule, treatment of internal displacement is not successful, excepting in case of strangulated inguinal, umbilical or abdominal hernia, which may be reduced by taxis, or by incision of the sac, enlargement of the hernial orifice and replacement of the constricted bowel-loop, if it be not already necrotic. In addition to the use of purgatives and enemas, attempts may also be made to detach from the rectum any connective tissue which may be adhering there, if such be the cause of the constipation ; or the sick animal may be moved into various positions, rolled over, led uphill and downhill—though usually without effect. The same may also be said of the former practice of administering large doses of mercury. The only remedy for many internal displacements, viz. :—laparotomy and manual rectification, can usually not be adopted, owing to the uncertainty of the diagnosis and the danger of an operation. It has, however, been carried out with success in isolated cases.

According to Jelkmann the diagnosis of a volvulus of the left layers of the colon should be positive, if, on inserting the left hand, immediately in front of the pelvic entrance near the fourth lumbar vertebra, the posterior mesenteric cord is felt to be tightly stretched, and in a diagonal position, instead of being perpendicular. It should be exceedingly sensi-

tive to the touch. If the twisted layers of the colon be tightly packed or distended, a second tensely strung cord will be found in the left region of the flank (bands of left inferior colon layer). According to Jelkmann this volvulus may be removed as follows :—Pass the left hand towards the left abdominal wall and push the left layers of the colon forwards and inwards. On reaching the region of the median line, raise the hand slowly, whereupon the upper layer of the colon, with the lower one, will sink over the hand into its normal position. By the upward movement of the hand the twisted loops of the rectum are also said to be lifted, so that both layers of the colon are able to fall back upon the left inferior abdominal wall. These statements have been confirmed by others. The Bavarian military veterinary report of 1891 states that Jelkmann's method had been applied with partial success in Bavaria twenty-five years earlier. The operation is facilitated by slightly raising the posterior parts of the horse.

4.—CONSTIPATION-COLIC CAUSED BY INVAGINATION OF THE INTESTINE.

Definition.—Invagination, or intussusception, consists in the intrusion of any intestinal part into the lumen of a neighbouring part. Most frequently, it is the ileum which is projected into the cæcum, and often, indeed, to a very considerable length ; or it consists in an invagination of the small intestine upon itself. Less frequently the cæcum is thrust bodily into the colon. The rarest cases are invaginations of the rectum into itself. In one case upon record, the small intestine had penetrated into the cæcum as far as the colon and rectum. The causes of invagination are generally to be sought in paralysis of the peripheral (distal) parts of the intestine, and penetration into the former of the more actively peristaltic central (proximal) portions. In one case the phenomena of invagination were noticed in a saddle horse immediately after jumping.

Symptoms.—Cases of invagination-colic are identical with those caused by internal displacement. The duration is, however, generally longer, because, in invagination, the whole lumen of the bowel is not usually blocked. A case is known in which, after ten days of diarrhœa and apparent improvement, death took place in 27 days. Animals with intussusception of the cæcum into the colon often live for a long time, and seem able to accustom themselves to this condition. In rare cases, a definite recovery is made by casting off the invaginated part of the bowels, and the growing together of the two ends of the intestine.

A positive *diagnosis* of invagination can only be made when necrotic, tubular portions of intestine are passed.

The *treatment* is consequently somewhat uncertain, and can, at best, only consist in flooding the intestines to procure easier passage, in the administration of purgatives, and in the introduction of large quantities of water into the rectum.

5.—CONSTIPATION-COLIC CAUSED BY NEW GROWTHS.

Anatomical Notes.—New growths in the intestinal canal are rare—and consequently seldom produce colic. They either narrow the lumen of the gut by affixing themselves to its side, or by compression from without, as, for instance, when situated on the mesentery. Thus, stomach-polypus may obstruct the duodenum and stretch it even to rupture. The lumen of the small intestine may be narrowed by fibromata, melano-sarcomata, myxo-sarcomata, leio-myomata and submucous lipomata, whereas in the large intestine and rectum the new growths are chiefly sarcomatous or cancerous. Lipomata occur rather frequently on the mesentery, where they cause bowel constriction or strangulation. Finally, stenosis of the intestine may be produced by abscesses on their walls, polypus in the rectum, proliferations of the intestinal mucous membrane resembling valves or pouches, tumours in the ovaries (ovarian cysts), as well as by phlegmonous inflammation of the connective tissue of the pelvis. The contents of the stomach usually lodge in front of the constriction, and cause expansion and compensatory thickening of the walls. The surface of the tumour frequently shows ulcerous degeneration.

Symptoms.—The symptoms of this rarer form of colic are chronic constipation with intermittent attacks of colic, which grow more and more intense. This condition may last for years, until finally, when the stenosis is more developed, the animal perishes from fæcal stasis. New growths in the rectum show themselves by the straining of the animal during evacuation, and the passing of blood-stained fæces. This may be confirmed by examination of the rectum.

Therapeutics.—The most important point is to keep the bowel contents in a liquefied condition by means of purgatives. When the new growths can be felt per rectum they may in some instances be removed by scraping, or if pedunculated by torsion. In all cases the rectum should be emptied manually

especially in spring and autumn. In breeding establishments, among well-bred finer-skinned horses, this is by far the commonest form of colic, as also among cavalry horses, with which —except in times of war or manœuvres—irregular feeding does not often occur.

Symptoms.—The pain arising in colic independently of all intestinal changes is probably traceable to compression of the sensitive nerves of the bowels by contraction of their muscular walls. Some believe it to be the same as rheumatic muscular pain, others ascribe it to the movements of intestinal gas. In spasmodic colic this pain is usually *rather violent, if not of very long duration.* It appears quite suddenly and in spasms, soon to abate and then reappear again with increased intensity. This intermittent, spasmodic course is very characteristic of the condition. In addition to this, in contrast to all other forms of colic, stoppage and the cessation of peristaltic action may be absent in cases of spasmodic colic, and the bowels may be unusually active and noisy, ending even in diarrhœa, owing to their spasmodic contraction. The so-called "spasmodic rumbling" is very loud as compared with other kinds of colic. It sounds as though water drops were falling from a certain height upon a thin, ringing metal plate, and can be explained by the difference of tension of the gases in the several bowel sections, causing the gas to gush with small explosions from the highly charged loops into those less densely filled. Spasmodic colic generally passes off after a few hours, sometimes even in from ¼ to ½ an hour. When it lasts more than 12 hours, it may be followed by serious complications, and especially volvulus.

Differential Diagnosis.—Here we have especially to guard against confounding spasmodic colic with the embolic form. Among the large number of so-called nervous or rheumatic colic cases which recover, some are certainly of embolic nature, as, owing to their similarly sudden and intermittent appearance and the inability of positive diagnosis by operation, an exact differentiation is impossible. But this confusion cannot arise to the extent asserted by Bollinger, seeing that spasmodic colic is supposed to exist only when previous chill can be proved, while in embolic or thrombotic colic the absence of all external cause is a point of first importance in forming an opinion.

Therapeutics.—In the treatment of spasmodic colic the so-called "anti-spasmodic" remedies have always played a chief part. Of these morphia stands first. Whilst the indications for its use are in all other kinds of colic principally symptomatic, they are here especially essential. In many cases, therefore, morphia alone suffices. It not only removes the spasmodic and painful contraction of the bowels, but also the serious danger of twisting of the intestines. The subcutaneous dose of hydrochlorate of morphia is 5 to 7 grains in a 4 per cent. watery solution. The smaller doses should be given to well-bred, and especially to thoroughbred horses, to guard against certain unpleasant by-effects, such as restlessness, over-excitement, and loss of appetite. The colic pains and bowel cramp usually subside quickly, so that a second injection is rarely necessary. The other nervine sedatives, such as belladonna, hyoscyamus, aconite, ether, chloroform, etc., have become superfluous since the introduction of morphia.

The usefulness of warm infusions made from camomile, peppermint, valerian and elder-flowers cannot be disputed, as, from the ethereal oil they contain, they have a certain undoubted sedative power. Most ethereal oils, owing to their evaporation in the intestinal canal, produce an anti-spasmodic effect on the surrounding organs, reducing their reflex excitability. The administration of ethereal oils in the form of infusions should be adopted rarely, because of the dangers which accompany drenching, and of their much slighter effect as compared with morphia. Physostigmine, arecoline, and barium chloride are contra-indicated.

For external treatment, we recommend vigorous rubbing down with turpentine and spirits of camphor (1 to 10–20), and warm coverings. Cold wrappings and enemas are not advisable, as they only increase the contraction of the bowel muscles. On the other hand, the application of hot water cloths to the abdomen and warm injections have a distinctly soothing effect. Finally, complete rest is much to be preferred to any movement or exercise.

V.—FLATULENT COLIC.

Etiology.—This form of colic is usually caused by the consumption of food tending to produce flatulence. The foodstuffs specially provocative of fermentation have been already mentioned in our remarks on the general causes of

colic. Moreover, windsuckers when they swallow too much air are sometimes attacked by this form of colic, and according to some authorities, horses may occasionally suffer from it after moving rapidly against the wind. The gases developed in flatulent colic are generally carburetted hydrogen and carbonic acid. Pinner found 49 per cent. of carburetted hydrogen, 8 per cent. carbonic acid, and 42 per cent. of nitrogen. Hydrogen and sulphuretted hydrogen are also present.

Symptoms.—The phenomena of flatulent colic consist in a very rapidly developing distension of the abdomen by the above-named gases. The abdominal walls are then arched like a barrel, are very tense to the touch, and, on being struck, the flanks emit a loud and clear sound, sometimes even a metallic and re-echoing ring. On applying the ear very loud tinkling noises are heard. As this inflation increases, so also does difficulty of breathing, and the animal becomes anxious and restless. The visible mucous membranes are much stained with red, sometimes bluish, the jugular veins are prominent, and the heart palpitates. The animal's gait becomes stiff, and it staggers in the hind-quarters. Its recovery is shown by abundant discharge of intestinal gases, and by the sinking of the abdomen to normal shape and size. A fatal result is caused sometimes by compression and œdema of the lungs ; at others, by blood poisoning with carbonic acid, or by apoplexy, and finally by rupture of the bowels or diaphragm. The course of the disease is always very rapid.

Flatulent colic is not difficult to *diagnose*. The gases which accumulate during other forms of colic (thrombo-embolic colic and that due to constipation) may be known by their slower and more gradual appearance and their longer persistence ; for flatulent colic is always rapid in its development.

Therapeutics.—In the treatment of wind-colic, the most important matter is the puncture of the intestine at the right moment, as this is the only certain method of removing the accumulated gases. It is usually made in the region of the right flank, in the centre of a triangle determined by the external angle of the ilium, the posterior edge of the last rib, and the ends of the costal processes of the lumbar vertebræ. First of all the skin and trochar must be disinfected, hair clipped off, and the perforation then made with a lancet or fleam, the point of the trochar being directed downwards and forwards

towards the left elbow. Then the wound should be closed with collodium or sticking plaster. But if the left flank happens to be more inflated, or the only one accessible, the puncture may be made there instead. Puncture from the rectum, as already suggested, has lately been strongly recommended.

In many cases of slight tympanites, physostigmine ($\frac{3}{4}$–$1\frac{1}{2}$ grs.), and arecoline ($\frac{1}{12}$–$\frac{3}{4}$ grs.) do good service. But where the bowels are very tightly stretched, both these remedies are not without danger, owing to the risk of bowel-rupture. Enemas of ether, as used largely in France (viz. : 4 to 6 drams of ether in one quart of water), are also sometimes useful in slighter cases of wind and cramp colic. Other remedies used against flatulent colic are of much less value.

Care must meanwhile be taken to assist the passage of flatus by cold, as well as stimulating enemas ; cold cloths and kneading of the abdomen. The administration of so-called "absorbent remedies" is not to be commended. Some of them, such as spirits of ammonia and sulphuretted potash, actually increase the amount of gas by the addition of ammonia and sulphuretted hydrogen ; while others—lime-water for instance—are scarcely able to retain their neutralising properties beyond the stomach.

VI.—COLIC DUE TO WORMS.

Etiology.—Among the parasites which may produce colic the first in order of importance is Ascaris megalocephala, then Tænia plicata, mamillana, and perfoliata, Spiroptera megastoma, Strongylus armatus, Scelerostomum tetracanthum, and finally Oxyuris mastigodes, as well as Gastrophilus equi and hæmorrhoidalis. They may cause colic in two ways—either by forming knotted balls which set up obstruction (this occurs with both round and tape-worms), or through traumatic injuries, which provoke inflammation, ulceration, and even perforation of the walls. In the latter connection, the gastrophilus must be specially mentioned, as, although generally innocuous, it may, in isolated cases, cause fatal colic. Ascarides may also produce multiple ulcers and violent inflammation of the intestinal mucous membrane, if present in very large numbers. Perforation of the intestine, with subsequent suppurative peritonitis, has also been caused by Tænia perfoliata. Moreover it must not be forgotten that intestinal worms, and especially nematodes, are often found on dissection, and care

must be taken not to incautiously connect their presence etiologically with a preceding colic.

Symptoms.—No special phenomena attend worm colic. Young animals are most liable to be affected, owing to their lesser power of resistance to the parasites. The colic is usually recurrent, but of slight intensity; with excitable mares, extraordinarily violent and frantic attacks of colic pain may arise, owing to the presence of gastrophilus hæmorrhoidalis in the rectum. The course of the complaint is generally chronic, in which digestive disturbances alternate with attacks of colic; but it may become acute if perforation of the stomach walls occur, or absolute stoppage of the lumen of the bowel, or in cases of intestinal inflammation. In these last named forms it is more fatal; otherwise the prognosis in worm colic is not so unfavourable.

Therapeutics.—These consist in the administration of anthelmintics. First of these is tartar emetic, which is given dissolved in drinking water, say 2 to 4 drs. per diem. Other remedies are—arsenic, powdered in single doses of 15–30 grains; turpentine, 2 to 4 oz. in emulsion; santonine (2 to 6 drs.), santonica (2 to 4 oz.), and areca nut (3 oz.). For the larvæ of gastrophilus in the rectum we recommend enemas of soapy water, as well as dilute solutions of petroleum, benzine or creosote, together with manual removal. Carbon bisulphide is recommended for larvæ of œstrus in the stomach; dose, 300 grs. (Perroncito and Bosso).

COLIC IN CATTLE.

I.—COLIC IN CATTLE CAUSED BY INVAGINATION OF THE BOWELS.

Etiology and Symptoms.—This is the most frequent cause of colic in cattle, and seems to occur oftener in cattle than in horses. The origin of intestinal invagination is supposed to lie in a previous chill, severe exertion in climbing a hill, and the more active peristalsis thereby produced. The consequences are very characteristic. The animals are suddenly seized, without any previous indigestion, by a more or less severe colic, which lasts from six to twelve hours, and then completely disappears, when the patient appears to have entirely recovered.

But loss of appetite, suppressed rumination and the passage of fæces, which are at first diarrhœic and later dry, all point to a more deeply seated malady. Soon persistent and very obstinate constipation, which defies all purgatives, sets in, during which, and only after much straining, small, tough, slimy lumps of fibrous masses, sometimes smeared with tar-like blood, are evacuated. The peristaltic sounds are at the same time quite suppressed, the abdomen is gradually inflated by the generation of gas in the intestines, and is sometimes sensitive to pressure over a certain area ; the pulse becomes feeble and very quick, while the temperature is out of all relation to this rapid pulse, the body feeling cold to the touch. The beast stands in dull apathy. Retching is noticed in isolated cases. On examining the rectum, one may sometimes find in the course of the bowel a longish, cylindrical and painful swelling.

Course.—This is rather slow. Generally the animals die, often without any death struggle, on the sixth to the ninth day, sometimes after about fourteen days, but seldom as early as the fourth. Occasionally the attacks recur several times at intervals of from eight to twelve days. Recovery happens in rare cases through necrotic amputation of the invaginated part, with peritonitic adhesion and growing together of the severed ends of the intestine. Tubular pieces of intestine have in such cases been found in the fæces.

Diagnosis.—It is easy to recognise invagination of the bowels in cattle. Sudden attacks of colic followed by unusual quietness, very obstinate constipation, evacuation (with much straining) of bloody slime, absence of the sounds of digestion, severe general derangement, local sensitiveness of abdomen (especially on the right), the growth of a painful swelling on the course of the intestine, and the duration of these conditions for several days, with gradual increase of debility, are all points to aid in diagnosis. When exploring the rectum care must be taken not to mistake the left kidney for an abnormal tumour, as it normally projects some distance into the abdominal cavity (Gautier). When symptoms of colic appear in cows in calf, the vagina and rectum should be examined for signs of uterine displacement (Albrecht).

Therapeutics.—The treatment is chiefly surgical. The administration of purgatives is futile, so that no time should be wasted in giving them. Attempts to loosen the intussus-

ception by simple physical means, by repeatedly driving the sick beast up and down hill, seldom succeed. The operation consists in opening the abdominal cavity at the right flank, seeking for the invaginated spot (very difficult in cases of pregnancy), and separation by hand of the invaginated parts. If this does not succeed, then the invaginated portion of bowel must be cut off, and the ends joined by enteroraphy (Meyer, Taccoen, Schmidt, Guittard, etc.). In performing the operation it should be remembered that, after the severed ends of the bowel are folded in, peritoneum is sewn to peritoneum and not mucous membrane to mucous membrane.

2.—COLIC IN CATTLE CAUSED BY STRANGULATION OF THE INTESTINES.

Etiology.—Apart from the occasional strangulation of the bowels in perforations of the omentum, mesentery, diaphragm, broad uterine ligament, etc., bowel strangulations are most commonly met with in cattle in the so-called pelvic hernia or gut-tie. As is well known, this is traced to a peculiar method of castration, namely, traction on the spermatic cords. Injurious results from this method may follow in three ways—either a rent is formed in the peritoneal fold of the spermatic cord which runs along the edge of the pelvic entrance, into which an intestinal loop insinuates itself from before backwards, and becomes strangulated. Or by the sudden rupture of the spermatic cord the proximal end is jerked backwards into the abdominal cavity, and there twists round the intestines, producing the so-called entanglement. In either case a strangulation of the intestines is caused. Less frequently the stump of the spermatic cord adheres to the wall of the abdomen, and the cleft thereby formed is sometimes a cause of imprisonment of bowel.

Symptoms.—These are almost identical with those described in invagination of the bowels. The duration is also about the same, being six to eight days. For diagnosis the discovery of a doughy and painful swelling, in size between that of an apple and the fist, situated on the edge of the pelvic orifice, a hand-breadth from the internal right abdominal ring, is of first importance. The spermatic cord will also be found tensely strung.

Treatment.—This is purely surgical. The imprisoned bowel is either freed by taxis through the rectum, pressing the

loop forwards and downwards, and pulling the tightened spermatic cord from its position; or an incision is made in the flank, and the imprisoned part set free by the inserted hand. Leading the animal down hill, throwing him upon his back, and massaging the abdomen are also measures which commend themselves.

3.—OTHER FORMS OF COLIC IN CATTLE.

Colic rarely occurs in cattle, except from the two causes named, and from inflammation of the bowels. (See chapter on Gastro-Intestinal Inflammation.) As with horses, it may be developed by over-feeding, chill, stoppage of the bowels, new growths, fæcal matter, hair-balls, potatoes, the swallowing of after-birth, volvulus, stenosis of the intestine by congenital semi-circular valves or new growths (lipomata), prolapse of the liver through clefts of the diaphragm, gall-stones, intestinal catarrh, etc. But the attacks are generally not so severe; only rarely are plunging, rolling on the back, bellowing or leaping into the manger to be seen. The animal is generally restless, stamps, strikes its foot against its belly, lashes its tail, and frequently looks backward. The treatment consists, as with horses, in the use of physostigmine (1½ grs.), and of laxatives (sodium sulphate, 1 to 2 lbs.; aloes, 1 to 2 oz.; castor oil, 1 pint mixed with rape-seed oil or poppy-seed oil, 1 quart; croton oil, 15–25 drops), together with enemas and external friction. According to Guittard, the colic arising from the displacement of an over-loaded paunch towards the pelvic basin (pelvic hernia) in young steers should be manipulated by the hand from the rectum. Leimer found that in simple colic creolin given in warm milk was attended by the best results. Spören cured a case of stoppage of the small intestine (potatoes) by laparotomy.

Constipation.—Simple constipation, unaccompanied by colic, may be traced to the same causes as those of colic. Such are: over-feeding; feeding upon unsuitable fodder (such as green oak-leaves); stenosis of the bowels; intestinal stricture, especially stricture of the colon arising from abscesses and new growths; and impaction of fæces and also coprostasis. In new-born calves, an imperforate anus is a frequent cause of stoppage.

COLIC IN DOGS.

Etiology and Symptoms.—Serious colic very rarely occurs in dogs. (Among 70,000 sick dogs treated in the Berlin

dog-hospital during nine years, only three cases were really severe colic.) Violent colic is sometimes present in distension of the stomach (Kitt, Wallmann, Jensen). In other cases, symptoms of colic are absent, although the stomach may be very much inflated, and there may be retching (Cadéac, Bouvicini). On the other hand, we not unfrequently observe symptoms of restlessness, proceeding from pain in the abdominal canal, to which the name of colic might be given. The causes are : cold, accumulations of hard food, especially of bones, obstruction of the bowels by stones and other foreign bodies, intestinal parasites, and especially invagination of the bowels. Stress must be laid upon the fact that, although these things *may* cause colic they do not necessarily do so, and that genuine colic phenomena are so slight that one rarely hears of colic among dogs. Usually obstruction of the bowels by foreign substances, intestinal worms and fæcal matter, bowel invagination as well as helminthiasis, run their course without any colic. The most frequent form relatively is one caused by worms (*Tænia echinococcus*), in which, according to Roell, even maniacal attacks may arise. The colic symptoms consist in excitement, whining, restless running to and fro, frequent lying down and curling up. They seldom last long.

Treatment will depend on the cause. The following are given for the purpose of alleviating pain :—Morphia-injections ($\frac{1}{4}$–$1\frac{1}{2}$ grs.) ; opium in the form of Dover's powders (7 to 30 grs.) ; or tincture of opium (20–40 drops) with mucilage. Enemas may also be given, and hydropathic wrappings placed round the abdomen. Foreign substances in the bowels, volvulus of the stomach, and bowel-invagination can only be satisfactorily dealt with by operation.

COLIC IN SWINE.

Etiology and Symptoms.—Colic in swine is generally traceable to overfeeding with bad and indigestible food, bran, fermenting refuse, beer-grains and to the eating of sand (sand-hunger). Cold also and the presence of intestinal worms (*Ascaris lumbricoides* and *Echinorhynchus gigas*) may produce colic. Finally, every inflammation of stomach or bowels—such as poisoning or invagination of the bowels—produces colicky pains. The animals show great unrest, arch their backs, lie down, frequently groan and squeal, stretch themselves on the

ground and manifest muscular contortions and cramp. They also cease to take food, and evacuation is retarded.

Treatment.—This is restricted to the administration of purgatives, anodyne electuaries, enemas, friction of the belly, etc. At the same time, warm shelter and soft litter must be provided. Calomel (½ to 1 dr.) is recommended as an aperient.

Constipation Colic in Elephants.—Munn cured severe colic with tympanites in an Indian elephant by water clysters injected by means of a hand fire-engine.

ULCERATION OF THE STOMACH AND INTESTINES

General Notes.—This is seldom seen as an independent disease in domestic animals. It occurs chiefly as a secondary phenomenon in the course of other diseases, such as rabies, rinderpest, malignant catarrhal fever, petechial fever in horses, dysentery, gastro-enteritis, gastro-intestinal catarrh, poisoning by corrosive substances, or injury caused by swallowing foreign bodies, and the larvæ of Gastrophilus, Spiroptera sanguinolenta, etc. These ulcers may, however, under certain circumstances, produce a definite aspect of disease. There are two kinds to be considered: 1st, ulcers arising from inflammation, particularly the so-called catarrhal ulcers and hæmorrhagic erosions; and, 2nd, the so-called peptic, round ulcers of the stomach and duodenum.

Etiology and Pathogenesis.—1. *Ulcers of inflammatory origin* develop after inflammation of the mucous membrane of the stomach and intestine, when the inflammatory infiltration has been very severe or accompanied by excessive hæmorrhage into the tissue of the mucous membrane. In such cases, even after the inflammation has abated, permanent ulcerous losses of substance may accrue to the membrane. These occur at various points in the stomach, but in the intestine they are usually found on the lymph follicles (follicular ulcers). Most frequently they form so-called catarrhal ulcers in the course of acute and chronic gastro-intestinal catarrh. Even after injury and partial cicatrization of the mucous membrane, ulcers may remain behind in any part of the intestinal canal.

2. *The so-called round peptic ulcer* occurs in the stomach and duodenum only as far as the acid gastric juice reaches. The opinion used to be held that it arose—in contradistinction to

that produced by inflammation—from a local self-digestion of the stomach. The sources of this gastro-malacia were sought either in local disturbance of the circulation, which hindered the regular neutralisation of the acid gastric juices by the alkaline blood, whereby the mucous membrane was exposed to its acid action, limited stoppage of the vessels of the mucous membrane by thrombi and emboli; fatty, sclerotic and amyloid degeneration of the vessels; local vascular spasms. Or they were found in an abnormal acidity of the gastric juice, which changed the slightest abrasions of the mucous membrane, that would under ordinary conditions speedily heal, into ulcers. General anæmia of the body, as well as venous hyperæmia (due to over-charging) of the mucous membrane were supposed to have precisely the same effect. Here, too, the mucous membrane, being weakened by deficient supply of arterial blood, was supposed to be unable, if injured, to withstand the action of the gastric juices. Nowadays the ulcers are believed to originate in the mucous membrane by specific wound-infection (inflammation followed by necrosis). In what manner ulcers of the stomach may be caused by general combustion, or by poisons contained in the blood, is a question which remains to be further investigated. Experiments show that they may be produced by injury of the spinal cord, of the corpora quadrigemina and of the medulla oblongata.

Peptic Ulcers of the Abomasum in Calves.—Of this R. Ostertag has described six cases. Most of these ulcers were perforating, and the stomachs were all in an intensely catarrhal condition. With respect to the pathogenesis of the ulcers, examination, both macroscopic and microscopic, showed that thrombosis took no part in it, but that they arose from circulatory disturbance was clearly indicated by their form. Ostertag establishes, moreover, the connection between the stomach-catarrh and the ulcers, by calling attention to the catarrhal process combined with nutritive derangement. Rasmussen and Kitt have described similar ulcers in calves.

Anatomical Conditions.—1. *Catarrhal ulcers of the Stomach* usually follow severe hyperæmia of the mucous membrane; and so-called hæmorrhagic erosions, catarrhal inflammations of hæmorrhagic nature. These erosions are most frequently found in the right half of the stomach, near the pylorus, and form small, round, superficial defects of the mucous membrane, with sharp, smooth edges and uneven base, generally covered with blood. Catarrhal ulcers usually arise in the bowels from

previous follicular ulcerations, which in their turn have been preceded by extreme cellular infiltration of the lymph follicles, with subsequent destruction of the tissue, and present the appearance of small, scattered, pan-shaped ulcers. As the disease advances they change their aspect. First, they grow larger—the size of half-a-crown or the palm of the hand; later, the ulcerous action is no longer confined to the mucous membrane, but attacks also the submucous and even the muscular coats, when bleeding from the eroded vessels of the bowels often supervenes. Lastly, even the serous covering may be attacked, which gives rise to peritonitic adhesions and perforation. Along with this progressive course a healing process is also going on. Many ulcers heal completely, leaving radiating scars. In other larger ones the edge heals over, grows thicker, and often as hard as cartilage, while the neighbouring mucous membrane arranges itself in striated folds, and the submucous and muscular layers are replaced by firm, fibrous connective tissue. Signs of chronic catarrh are also present.

2. *Peptic ulcers* have been hitherto but little observed. They occur only in the stomach and at the commencement of the duodenum, and are thereby distinguishable from the others. They are usually quite regular in shape, roundish or elliptical, have sharp, smooth edges, appearing as if punched out; and generally occur singly. They heal with a radiating scar, and may produce peritonitic adhesion, as well as internal bleeding and perforation.

Symptoms.—It is impossible clinically to differentiate between these two forms of ulcer; their symptoms being similar and often very characteristic. The aspect of the disease is frequently identical with that of chronic gastro-intestinal catarrh. Thus Siedamgrotzky noted in a horse only gradual wasting and exhaustion; and Grad and Zippelius in a cow the symptoms of chronic catarrh of the abomasum, viz.: chronic tympanites, constipation, anæmia, and general disturbance of nutrition. Sometimes more striking symptoms are seen, from which the presence of an ulcer can with some probability be diagnosed. These consist in obstinate vomition, signs of colic, hæmatemesis, and the passage of blood with the fæces. In horses periodic attacks of colic, recurring at certain times after meals, have been chiefly noticed, with gradual falling off in appetite and even the vomiting of blood-stained matter. In cattle Gotteswinter observed, along with anæmia and uncertain gastric

disturbances, that the dung was black and tar-like, indicating the presence of ulcers in stomach or bowels, which was confirmed on slaughter. Wesikopf noted several cases of rupture of the stomach in horses, caused by ulcers in the stomach. According to Rasmussen the presence of perforating ulcers in the abomasum of calves may be presumed (in life) by the animals growing restless after transport, throwing themselves down, lying languidly upon the ground, puffing and staggering and tumbling when lifted up. The course of the sickness caused by ulcers in stomach or bowels is usually very protracted. Recovery is possible, but the termination of a case which admits of positive diagnosis is usually fatal, death resulting from loss of blood, from perforative peritonitis, or, finally, from gradual exhaustion and wasting.

Differential Diagnosis.—Owing to the difficulty of precise diagnosis, and the usual lack of pathological indications, this complaint is often confounded with others, especially with gastro-intestinal catarrh, which is easily understood, seeing the close connection between the two. But even when the characteristic symptoms are present, such as vomiting and hæmorrhage of stomach and bowels, it is not always easy to distinguish this disease in cattle from that produced by the presence in the stomach of foreign substances, as precisely similar symptoms occur. In such a case reliance must be placed mainly upon the presence of pain in the second stomach, which is generally absent in cases of ulcer in the abomasum.

Treatment.—The treatment for ulcers in stomach or bowels must be first of all purely dietetic. Its chief object should be to guard the ulcer against irritation by coarse or indigestible food. Zippelius, therefore, recommends for cattle liquid and well-cooked nourishment only, and entire avoidance of substances which readily ferment, such as brewers' grains; for horses meal and bran-mash; for dogs milk and raw eggs, with finely-chopped meat. Of medical remedies artificial Carlsbad salt is the best. It not only neutralises the excessive acidity of the stomach, but ensures its disinfection and rapid emptying. Use either the official preparation or prescribe it in suitable mixture, say—sulphate of sodium, 2½ drs.; chloride odium, 5 drs.; bicarbonate of soda, 75 grs.; giving to a horse t to cattle 3 oz., and dogs ½–1 dr. per dose. Spirits of salt ontra-indicated. Zippelius remarks on this point that

intestinal ulceration in cattle is probably that serious form of indigestion in which spirits of salt (hydrochloric acid) acts as a poison! On the other hand, in obstinate cases one may make a trial of subnitrate of bismuth (2 to 8 grs. for a dog), or of nitrate of silver. In hæmorrhage of the bowels prescribe styptic remedies (tannin and preparations of iron or lead). To check persistent vomiting in dogs, use the narcotics already advised for catarrh of the stomach, viz.: opium, cocaine and bromide of potash, also creosote, creolin, tincture of iodine, fragments of ice, etc.

HÆMORRHAGE OF THE STOMACH AND INTESTINES.

Etiology.—This may arise from many causes, which are not always easy to identify. In spite of the fact that such hæmorrhage is only a symptom of some other disease, it is wise to treat it carefully and independently. It is understood, of course, that we speak only of cases in which a large discharge of blood is noted.

1. In many cases hæmorrhage is caused by mechanical or chemical injury to the gastro-intestinal mucous membrane. Such injury may arise from nails, bone-splinters or glass, fish-bones, sharp pine-needles, etc.; also from entozoa (larvæ of gastrophilus, Tænia echinococcus, Echinorrhynchus gigas); from irritants, such as undiluted tartar emetic; and, lastly, from concussion of the stomach produced by a kick, a fall or over-exertion.

2. Ulceration of stomach or intestines, especially the peptic form, may produce bleeding by opening the blood vessels.

3. Hæmorrhage very often occurs in the course of acute inflammation of the mucous membrane, as, *e.g.*, in the various forms of enteritis and in the distemper of dogs.

4. Venous congestion of the mucous membrane of the stomach, or bowels is occasionally followed by hæmorrhage. Such congestion ensues upon displacement of the bowels, as well as disorders of the heart, lungs or liver (valvular defects, emphysema of the lungs, induration of the lungs, cirrhosis of the liver, fatty degeneration of the same, or thrombosis of the portal vein).

5. Diseases of hæmorrhagic nature are sometimes accompanied by intestinal hæmorrhage. Among these are certain forms of anthrax (hæmorrhagic intestinal anthrax), petechial fever in horses, septicæmia, and septicæmic puerperal fever in cattle.

6. Hæmorrhage of the rectum arises from injuries, tumours and hæmorrhoids. The latter occur as diffuse or nodular varicose enlargements of the hæmorrhoidal veins, partly in the submucous (so-called internal hæmorrhoids), partly in the subcutaneous tissue outside the anus (so-called external hæmorrhoids). They are most frequent in swine, rarer in dogs, horses and cattle. They arise after general congestion of the circulation (heart, lung or liver complaints) through insufficient emptying of the hæmorrhoidal veins, which are furthest from the heart of all the veins in the abdominal cavity. In pigs the evil is possibly also hereditary. Over-feeding with rich, indigestible or irritating food is also sometimes the source, or lack of exercise, excessive use of drastic purgatives, etc. Enzootic inflammation is occasionally found as the cause of hæmorrhage of the rectum in cattle. The so-called " back or loin blood " was formerly attributed by Rychner (Bujatrik) to hæmorrhoids, with which Spinola declared it to be related; whereas Haubner-Siedamgrotzky considered that it was caused by sharp, irritating and too highly seasoned food, or by over-exertion, which produced inflammation of the mucous membrane of the rectum. According to this, " loin-blood " is no other than a hæmorrhagic proctitis. In other cases, anthrax is the cause.

7. Vogel mentions a peculiar kind of hæmorrhage of the stomach. An aneurism of the abdominal aorta in a dog had grown fast to the stomach, and, on rupturing, emptied itself into the stomach, causing speedy death. A similar case was noticed in a mare by Labat and Cadéac, in which an aneurism of the aorta had emptied itself into the colon; and there was another of a horse which bled to death as the result of an intestinal angioma.

Symptoms.—The most prominent sign of stomachic hæmorrhage is the vomiting of blood (hæmatemesis). A large amount of lumpy, dark-coloured blood mingled with food is discharged during repeated attacks. This symptom may, however, be absent, in which case the hæmorrhage of the stomach is hardly distinguishable from that of the bowels. The latter is recognisable by the passage of fæces, which are either mixed with blood, or discoloured and tarry. At the same time, the signs of internal bleeding or chronic anæmia are present, and may lead later to hydræmia and serous transudation into the cavities of the body.

The phenomena of hæmorrhagic inflammation of the rectum

in cattle are sudden feverish attacks, stiffness of attitude, sensitiveness about the croup, straining to evacuate, which, however, only results in the passage of a few dry, blood-mingled fæces ; also enhanced temperature, puffy swelling of the mucous membrane of the rectum, so that the hand, on being inserted, becomes bloody. Recovery usually follows after from three to five days, but where the inflammation is intense, the end may be fatal.

Utz noticed in a horse, along with two clumps of piles the size of a walnut, frequently recurrent colic, with discharge from the anus of dark, fluid blood and occasional dark blood clots, followed later by anæmia and hydræmia combined with bleeding of the skin (so-called bloody sweat), which eventually caused death.

Differential Diagnosis.—It is not difficult to differentiate hæmorrhages due to gastro-enteritis from those due to proctitis or hæmorrhoids, but it is frequently difficult and even impossible to diagnose or locate with certainty such conditions as ulceration, irritation by foreign bodies, chronic congestion or hyperæmia. It is often important, in cases of discharge of blood from the mouth, to decide whether one has to deal with hæmorrhage of the lungs or of the stomach. The latter is indicated by previous and subsequent gastric disorders, by distinct retchings, disgorgement of the contents of the stomach, the appearance of tar-like fæces, absence of cough and soundness of the lungs. The condition of the vomited blood is also an indication, as in hæmatemesis it is mostly clotted, dark in colour and showing acid reaction owing to the presence of gastric juice. In contrast to this, the blood discharged in hæmorrhage of the lungs is bright red, has an alkaline reaction, and, owing to admixture of air, is frothy. Both before and after the hæmorrhage, difficulty of breathing and coughing will be observed. The differentiation may often be very difficult, when, for example, the blood coughed up is swallowed and afterwards vomited, or when, in hæmatemesis violent coughing accompanies the retchings. Spinola mentions a case in which vomiting in a dog after swallowing a quantity of blood was mistaken for hæmatemesis. Finally, hæmorrhoids in dogs are often confounded with over-filling of the anal pouch and formation of abscesses therein.

Treatment.—The treatment of hæmorrhage of the stomach or bowels is best carried out by means of styptics.

To these belong cold water, small pieces of ice and astringents, viz.: alum, tannin, sulphate or perchloride of iron (dogs 3-5 drops in a pill), sugar of lead, nitrate of silver, opium, ergot of rye and extract of hydrastis (U. S. P.).

CANCER OF THE STOMACH.

The domestic animals, as compared with man, appear to be almost immune from cancer of the stomach. Its presence is, as a rule, only discovered upon *post-mortem* examination. Among 70,000 dogs treated at the Dogs' Home, Berlin, in the years 1886 to 1894, one case only of cancer was observed, and among horses none have ever been recorded in Germany. It is impossible, therefore, for obvious reasons, to give a clinical *picture of the condition*. The following cases have been recorded: In making the *post-mortem* examination of a horse which had shown symptoms of dull colic accompanied by wasting, Petit and Fayet found an ulcerous growth, as large as two fists and shaped like a cauliflower, in the cardiac end of the stomach. McFadyean observed a case of primary carcinoma in the stomach of a pony; there were numerous metastases from the stomach to the liver, peritoneum and mesentery. Dürbeck and Cadiot have reported upon similar cases in horses. Eberlein describes a case of primary cancer of the stomach in a dog which, owing to occlusion of the gall-ducts, gave rise to severe jaundice. Parascandolo is said to have successfully treated cancer of the stomach in a dog by resection (gastrectomy). The animal had, for twelve months, shown great emaciation, accompanied by vomiting, colic, blood in the fæces and constipation.

Cancer of the Bowels.—Meis and Parascandolo describe a case of cancer of the cæcum in a dog. The condition was accompanied by chronic emaciation, vomiting of bile, constipation, colic, collapse and general prostration. The diagnosis was confirmed by laparotomy, but the operation was unsuccessful.

GASTRO-ENTERITIS.

Classification.—Inflammation of the gastric and intestinal mucous membranes of the domestic animals may be variously classified. In this chapter we shall consider, first the primary and independent inflammations, not those which are secondary, and which arise in the course of other diseases, such as rinderpest, influenza, dysentery, swine-fever, rabies, petechial fever or displacement of the bowels. An attempt has been

made to divide gastro-enteritis, according to the chief seat, into an inflammation of the stomach "gastritis," and of the bowels "enteritis," and to subdivide the latter on the same principle into duodenitis, ileitis, typhilitis, colitis and proctitis. But in practice this can seldom be done, as both stomach and bowels are usually simultaneously affected. Rather might one take the degree of inflammation as a basis for classification and speak of parenchymatous, phlegmonous, suppurative, hæmorrhagic, croupy, diphtheritic or caseous inflammations. The lowest degree would be represented by gastro-intestinal catarrh. Even better would be an etiological classification into: 1—Inflammation caused by simple mechanico-thermal irritants; 2—that produced by poisons (toxic); 3—Infectious inflammation (dysentery); 4—embolic; 5—tubercular; 6—mycotic (produced by moulds), etc., etc. To speak of diffuse or circumscribed gastro-enteritis, of sporadic or enzoötic, of acute or chronic, is much too general a differentiation. For clinical purposes we advise a classification based partly upon the degree and partly upon the cause. We, therefore, distinguish the following as forms of gastro-enteritis which can be more or less well characterised as independent and individual diseases:—

1. Simple, Non-Toxic Gastro-Enteritis of various domestic animals.
2. Croupous Gastro-Enteritis, especially of cattle.
3. Mycotic Gastro-Enteritis (so-called Typhus).
4. Toxic Gastro-Enteritis.

I.—SIMPLE, NON-TOXIC GASTRO-ENTERITIS.

Etiology.—The causes of simple ordinary gastro-enteritis differ from those which produce acute gastro-intestinal catarrh only in degree. Among these are violent internal or external chill, scalding of the gastric mucous membrane by hot drink (*i.e.*, by giving calves their milk too hot), irritant food, foreign bodies, and intestinal worms, especially the spiroptera species (sp. megastoma in horses; sp. sanguinolenta in dogs; sp. strongylina in pigs, etc.). Hay-mites (Acarus fœnarius) have been cited as causing gastro-intestinal inflammation in horses, but probably incorrectly, as they are more or less present in all hay. With regard to gastro-enteritis due to coccidiæ and gregarines, compare the chapters on coccidian dysentery (bloody flux), coccidiosis of the intestines and liver, and also on diphtheria in birds.

Pathological Notes.—The symptoms of gastro-enteritis differ from those of acute catarrh only in intensity. But occasionally one form passes into the other and they become difficult to distinguish. In inflammation, however, we find hæmorrhage instead of hyperæmia, and parenchymatous, phlegmonous, serous, or suppurative infiltration of the mucous membrane, even necrosis, instead of superficial desquamative processes.

1. Examination of the stomach reveals considerable redness, either circumscribed or diffuse, and varying in shade from light to dark. The mucous membrane is dull and swollen, sometimes in folds and infiltrated with blood, or covered with petechial spots or larger dark-red patches. The surface of the membrane is sometimes smooth, at others covered with tough or thin, clear or blood-coloured slime, and again it reveals small, round or longish, superficial ulcers in greater or less number, whose edges are sharp and even, their base at times smooth and at others covered with blood coagulæ (hæmorrhagic erosion). These occur generally upon the folds and in the pyloric region of the stomach, and are most frequent in dogs and in the abomasum of cattle, where they are induced by hæmorrhagic infiltration of the mucous membrane, with consequent superficial necrosis. The surface of the membrane is, moreover, roughened by the swollen and fatty disintegrated peptic glands, which project in the form of granules. Sometimes the submucosa is infiltrated with œdematous or purulent fluid.

2. In the bowels the mucous membrane is also red, swollen and dull, usually to a limited extent, but sometimes all over, or in dots, tooth or star-shaped or like net-work, and at times is marked with light or dark-red spots, stripes or patches. It is also infiltrated with fluid, soft. tender and disintegrated to a pulp, or like tinder, occasionally covered with dry, discoloured gangrenous scabs, which are either firmly fixed, or have been partly loosened by suppuration, and may have caused complete perforation of the intestinal wall. The epithelium of the mucous membrane is frequently dislodged over a considerable area, and the tufts of villi are swollen and prominent, look larger, float plainly under water and give to the mucous membrane a velvety appearance. The solitary and agminated follicles are sometimes found unchanged, and at others swollen and surrounded by a patch of red, the solitary ones appear like nodules the size of a pin-head, the agminated ones larger and irregular. On disintegration of these, crater-shaped follicular ulcers remain. The contents of the bowels vary much in nature, being some-

times thin and watery, sometimes slimy and putrescent, at others clear, or milky, greyish or stained with blood, or mixed with epithelial flakes, coagulæ of fibrin, small lumps of pus or blood, or they may even consist of blood alone. The submucosa is either normal or highly injected and œdematous, gelatinous or hæmorrhagic and swollen, making the mucous membrane appear extremely thick and puffy. Thus, in the cæcum and colon of the horse it may be found several fingers thick over a considerable surface. The muscularis is usually only slightly affected by serous infiltration, unless it be perforated or deeply ulcerated. The serosa is injected, and often shows a coating, as well as peritonitic proliferations. Finally, the mesenteric glands are frequently infiltrated and swollen, and the liver and spleen very full of blood.

Symptoms.—The phenomena of simple sporadic gastro-enteritis either follow gastro-intestinal catarrh, or appear suddenly and independently, which is generally the case. Appetite and rumination are at first entirely suppressed. Swine and dogs often vomit frequently, and both horses and cattle are nauseated. The membrane of the mouth is very red, hot and dry, and in later stages intense thirst is developed, the satisfaction of which may cause increased vomiting, especially in dogs. To this are added, continuous and generally very violent symptoms of colic, which in dogs may amount to frenzy. The stomach and abdomen are painfully sensitive to touch, the latter tense and sometimes tympanitic. Peristalsis is suppressed, and obstinate constipation ensues, during which the animals pass at most a few hard, small balls of fæces (after much straining), which are covered with mucus or blood, or they pass pure blood (hæmorrhagic enteritis). In exceptional cases gangrenous fœtid sloughs of intestine are evacuated. It is not until near the fatal termination that the constipation changes to severe diarrhœa, when to the paralysis of the intestinal walls is added that of the sphincter ani. The urine frequently contains albumen and, in herbivora, acid phosphates. The severe gastric attacks are accompanied by strong fever. This is indicated by a very quick, small, hard pulse, great rise of body temperature, highly reddened mucous membranes, coldness of the extremities and sweating. In horses the pulse may be 80 to 100 beats a minute, and the internal temperature 104° to 106° F.; with the approach of death the temperature becomes sub-normal. The general sensibility is often very greatly dimi-

nished; the animal shows great fear and restlessness, or the excitement yields to a gradually increasing stupor and collapse, it sways and staggers and has occasionally convulsions and spasms. Towards the end the colic pains usually abate, and the horse expires with the appearances of coma.

The progress of simple gastro-enteritis is very rapid in the case of the horse, so that its duration seldom exceeds two or three days. The chronic form, characterised by recurrent attacks, usually lasts several weeks and terminates in death. It presents the same symptoms in the horse as thrombo-embolic enteritis. Gastro-enteritis occasionally occurs enzoötically.

Prognosis.—The disease is very serious and generally ends in death. Favourable appearances are—moderate symptoms of colic, low fever, especially a strong and steady pulse and regular evacuation. Unfavourable, on the contrary, are—very high fever, a much accelerated, small and wiry pulse, dyspnœa, obstinate constipation, sudden cessation of pain, accompanied by collapse and profuse diarrhœa. Death follows probably from necrosis of the bowels with consequent general sepsis, or from apoplexy of the brain, œdema of the lungs, intestinal hæmorrhage, or paralysis of the heart caused by shock.

Differential Diagnosis.—1. To distinguish gastro-enteritis from gastro-intestinal catarrh, one has to be guided chiefly by the intensity of the inflammatory symptoms: high fever, weak action of the heart, general condition, severe colics, and constipation.

2. In horses it is more difficult to distinguish from other forms of colic a gastro-enteritis which first develops as a so-called "Inflammatory Colic." In many cases it is impossible during lifetime, and only a *post-mortem* examination can supply a correct diagnosis. A further great difficulty lies in the fact that many a colic terminates in enteritis, and a differential diagnosis should in every case be at least attempted. Guiding indications are: high fever, rapid, small pulse, intermittent colicky pains, general debility, prolonged constipation, painful sensitiveness of the abdomen to pressure, and the previous history of the case. Occasionally a diagnosis may be made by exclusion.

3. From the remaining forms of gastro-enteritis the foregoing may, in general, be distinguished by observing the following signs:—In the toxic form the previous history of

the patient and the characteristic signs of certain poisons; in mycotic cases the peculiar nervous phenomena; in the croupy form the passage of fibrinous membranes and its longer duration; in infectious dysentery its enzoötic spreading and the prevalence of diarrhœa.

4. A primary gastro-enteritis may be differentiated from anthrax, rinderpest, etc., by careful examination and watching the progress of the disease.

Therapeutics.—After the removal of the exciting causes, the treatment of simple gastro-enteritis demands careful watching and regulation of diet. At first complete abstinence is to be advised, followed by light, digestible food. Harm is occasionally done by prematurely giving food difficult of digestion. The water must be tepid. Dogs should have as little water as possible, as drinking produces vomiting.

The further treatment begins with restoration of activity to the skin by friction, rubbing the abdomen with spirits of camphor, turpentine, or mustard oil, and applying wet cloths. Inwardly administer soothing remedies, especially mucilaginous decoctions and oil-emulsions, such as linseed, fresh linseed-oil, barley-water and mucilage mixed with opium, tincture of opium, etc. If the colicky pains are violent, use injections of morphia. Caution is necessary in using drastic remedies against obstinate colic, owing to the risk of increased irritation of the mucous membranes. Use, therefore, only the mildest purgatives, such as castor oil in emulsions, Glauber's salt and sulphate of magnesia, as well as calomel, the latter often in combination with opium.

As examples, the following recipes may be useful:—

For the horse: An electuary composed of powdered opium, 2½ drams; calomel, 30 grains; powdered marshmallow, 3 ounces; distilled water q.s. (one dose).

For the dog: Tincture of opium, 30 to 75 minims; mucilage of gum arabic, 5 ounces; water, 5 ounces (dessert-spoonful doses).

In great weakness or collapse prescribe camphor, ether, wine, coffee, caffeine, hyoscyamus, etc.

2.—CROUPOUS ENTERITIS.

Occurrence.—Croupous enteritis is most common in cattle, far more so than is generally supposed. It is rare in the horse,

and the carnivora (cats), and very rare among sheep. It is sporadic in the ox and has always a chronic course. In the other animals it is also generally sporadic, but it may occur in either acute or sub-acute forms.

Etiology.—The causes of croupous enteritis in cattle are still very obscure. Cold is generally believed to exert some influence on its production, particularly as it is common in the spring and autumn. Irritating and indigestible foods are frequently responsible; hay with a large admixture of aromatic herbs; overfeeding with chaff. In some cases it has been traced to injudicious use of drastic purgatives. Other drugs, such as spirits of camphor, cantharides, etc., may also produce croupous enteritis in cattle. The reason why this complaint is chiefly noticed among cattle is that they are supposed to be naturally predisposed to it, which fact may rest on their blood containing much fibrin and albumen, with a tendency to fibrinous excretions. This is especially marked in young and well-nourished animals, and in those advanced in pregnancy, which suffer from intestinal croup with great frequency as compared with others. Perhaps, also, the lymphatic constitution peculiar to cattle may have some connection with it, one consequence of which, for example, is that in inflammation of the lungs enormous plastic exudations of that organ readily occur.

In horses constipation appears sometimes to cause intestinal croup. A similar condition may be caused by tape-worms in dogs and cats. A typical form of croupous enteritis in cats appears to be due to the presence of the bacillus coli (Zschokke). In many cases the cause remains unknown.

Pathological Notes.—The seat of croupous inflammation in cattle is either the small intestine or the colon; in horses, usually the small intestine. The mucous membrane presents catarrhal or inflammatory changes in different degrees, and is covered to a greater or less extent with greyish-yellow croupy matter, more or less easily detachable, beneath which the membranous tissue is very red, soft and friable and infiltrated with blood and pus. This croupous matter sometimes forms itself in cattle into stratified layers, and at others it is tubular, cord-like, or cylindrical, and either solid or hollow and filled with food. Occasionally it consists of several tubes, one within the other, which can be drawn out, and between which particles of food may be found. The length of these tubes varies considerably;

at times they are only short, but occasionally measure as much as ten yards. Microscopic examination reveals an amorphous structure of fibrinous matter, with embedded, fatty-degenerated epithelial cells, and white blood-corpuscles. The contents of the intestine consist either solely of this croupy matter, or in addition a thin, fluid substance, often smelling very badly and discoloured with clotted coagulæ. On closer examination the tubular glands of the intestine are also found to be filled with fibrinous exuded matter.

Symptoms.—The symptoms of croupous enteritis in cattle are much milder than those of other forms of enteritis. Occasionally no marked phenomena are noticeable; at other times they only appear in connection with the expulsion of croupy matter. Usually the signs which precede the evacuation of the above-named fibrinous formation are merely those of intestinal catarrh or of chronic enteritis. Disturbance of appetite and of rumination are noticed, with slight colic, constipation and low fever. After a few days the croupy discharge begins, combined generally with severe diarrhœa and passage of thin, fœtid, greyish-brown fæces, mixed with fibrinous shreds and occasionally infiltrated with clots of blood or pus. This evacuation is usually followed by improvement, often by extremely rapid recovery, so that the entire duration may in general be from six to eight days. In exceptional cases the disease in cattle may be more severe. Thus Festal records an acute croupy enteritis in the ox, which commenced with frequent colic, obstinate constipation, high inflammatory fever and tottering gait, in which, after abatement of the colicky symptoms, but with continuous constipation, membranous masses were squeezed out, followed six or seven days later by an ichorous, evil-smelling fluid. Then came improvement, or else death ensued after general increase of debility and formation of peritonitis. (Duration, from eight to twenty days).

Croupous enteritis generally manifests itself in horses by symptoms of persistent indigestion-colic. Prietsch reports in one case maniacal signs (kicking, biting, etc.). Sometimes these colicky symptoms are lacking, and the ailment proceeds essentially as a feverish affection of the bowels, with discharge of soft, fœtid fæces, mingled with flaky, clotted or stringy, croupy matter. In other animals the appearances resemble those of simple gastro-enteritis, but are less severe.

Croupous enteritis in cats begins with vomiting and diarrhœa and ends fatally within three days, frequently giving rise to a suspicion of poisoning (Zschokke).

Prognosis.—This is rather favourable for cattle. In rare cases, danger may arise from complete stoppage of a bowel-section by croupy matter. In other animals, especially horses and cats, the course is likely to be more serious.

Differential Diagnosis.—The passage of ropy or tubular croup-substance through the anus, which alone renders diagnosis of bowel-croup possible, may lead to error, especially with laymen. Thus these bands have often been mistaken for snakes or tape-worms, for intestines of smaller animals, which have been swallowed, or for intestinal loops of the patient itself. But a careful examination of these croupy cylinders, their greyish-white colour, homogeneous structure, the absence of mesenteric adhesions, or of any definite arrangement of blood-vessels, will easily prevent their being mistaken for intestines. In horses, slime-covered dung-balls, such as are often seen in ordinary constipation or catarrhal proctitis, must not be confounded with the products of bowel-croup. And in carnivora also the same mistake must not be made in the fæces containing mucus or half-digested connective tissue-like membrane, which proceeds from the food.

Therapeutics.—The treatment of croupous enteritis is not essentially different from that of the simple form. Solvent and liquefying remedies are to be preferred to emollient ones. The neutral salts, and particularly the alkalies, whose object is to dissolve the croupy coagulæ (bicarbonate and sulphate alkalies) are useful. These salts may also be administered in diluted form in enemas, particularly for dogs (a 1 per cent. solution of common salt, soda or potash).

3.—MYCOTIC GASTRO-ENTERITIS.

(Infectious, septic, septiform, typhoid gastro-enteritis ; intestinal mycosis ; mycosis or sepsis intestinalis ; enteric fever ; and poisoning by meat, fungi, or sausages.)

Definition.—All the above-named conditions are included under the title of mycotic gastro-enteritis. From the etio-

logical point of view they are similar, all being caused by the irritation of some injurious material, affecting first the intestinal canal and secondly the whole system. The active principles must be regarded as operating in a two-fold manner: first, *septic infection*, that is an invasion of micro-organisms (bacteria, micrococci); and secondly, as a *septic intoxication or poisoning*, due to the absorption of the chemical products of those micro-organisms. These chemical substances, the result of bacterial metabolism, are called "Ptomaines," "Toxines," "Corpse Alkaloids," etc.

Mycotic gastro-enteritis may therefore be more exactly defined as an intestinal affection, accompanied by general indisposition, and resulting from consumption of decayed foods, partly through the invasion of pathogenic microbes and partly through absorption of poisonous ptomaines. It lies midway between an infectious disease and toxic gastro-enteritis, possessing features common to both. There is a distinct connection between meat-poisoning and septicæmia on the one hand, and the poisonings due to fungi and acrid narcotics on the other.

The fact that mycotic gastro-enteritis is distinguished not only by genuine gastric disturbances, but also by grave general symptoms, such as great debility, cerebral disturbance and a rapid course, led in earlier times to its being called "typhoid" gastro-enteritis, or "bowel-typhus." It was erroneously believed to be identical, or at least analogous, to enteric fever in man.

In the following we describe, first, such mycotic gastro-enteritis as is caused in carnivora, omnivora and birds by the consumption of decomposed flesh; and then the similar ailments noticed in the herbivora after eating fungous vegetable matter (mould-fungi, uridineæ, or rust and smut-fungi). With regard to the latter we must observe that the kind of fungus cannot always be exactly determined, and that there are outbreaks which offer the typical aspects of fungous poisoning, although we are unable to prove that the fungi are the direct cause of illness. Sometimes, too, in cases of fungus poisoning, all signs of inflammation in the gastro-intestinal mucous membrane are absent, because the poison passes directly into the blood, without injury to that membrane, producing essentially nervous symptoms.

I.—MYCOTIC GASTRO-ENTERITIS OF CARNIVORA, OMNIVORA, AND BIRDS AFTER EATING DECOMPOSED FLESH. MEAT-POISONING.

Etiology.—Mycotic gastro-enteritis in our flesh-eating domestic animals is closely allied, both in origin and symptoms, to "meat-poisoning," as observed in man. Probably a great many of the cases formerly reported as "dog typhus" or "swine typhus" were mycotic gastro-enteritis. It is due to the consumption of meat in a more or less advanced state of decomposition, stale sausages, old herring-brine, or rotten cheese. It is commonly seen in butchers' and shepherds' dogs, and has also been noticed in fowls after eating bad meat. The actual cause of the illness is to be sought in the bacteria present in the meat as the result of putrefaction, and in the ptomaines to which they give rise. As little is usually known of the patient's recent history, the trouble is frequently mistaken for a case of mineral poisoning.

That the carnivora are very unequal in their susceptibility to mycotic poisoning is proved by various experiments with putrid meat. Semmer found that three swine died from the results of eating the flesh of a horse which had perished of septicæmia, while dogs and cats remained healthy. Colin made similar experiments and only observed slight diarrhœa. Lemke found that after feeding on flesh infected with black-quarter, three young dogs developed violent diarrhœa, while an older dog remained unaffected. The same variations as to the results of fungus poisoning have been observed among the herbivora.

Symptoms.—1. *In meat-poisoning* these consist in a sudden, very violent and often bloody diarrhœa, vomiting, intense thirst, high fever (104°–107° F.), extreme weakness and lassitude, followed quickly by collapse. Death often occurs in a few hours, usually within 24. Schindelka noticed hæmorrhage of the retina in two dogs, by ophthalmoscopic means.

2.—*In sausage-poisoning* (*botulismus, allantiasis*), the same symptoms are present, but characteristic indications of paralysis (vomiting, constipation, and less often diarrhœa weakness and paralysis) in the region of the 2–6 brain-nerves are also often seen, such, for instance, as affections of the sight, dilation of the pupils, ptosis (*i.e.*, paralysis of the upper eyelid), severe

tympanites (paralysis of the stomach), paralysis of the pharynx and of the larynx (aphony), and constipation (ditto of the bowels).

3.—*Fish-poisoning*, which occurs in swine after being fed with herring-brine, is characterised by spasms. The animals show cramp of the jaw, epileptic and tetanic convulsions, nystagmus, the phenomena of brain disorder, giddiness, symptoms resembling those of staggers, rotatory motions, inability to swallow, and amaurosis.

Post-Mortem Notes.—On examining the contents of stomach and bowels we will find half-digested, foul-smelling masses of flesh, chocolate coloured, bloody and of a mucous, fluid consistency; while the mucous membrane is swollen, considerably injected and hæmorrhagically infiltrated, the follicles, both solitary and agminated, as well as the mesenteric glands are all swollen. The blood is altered; the liver enlarged and rapidly decomposing; the spleen swollen and infiltrated with hæmorrhagic foci, while the muscular tissue of the heart is very friable.

The Etiology of Meat Poisoning.—The cases of meat-poisoning, which are of frequent occurrence in man, generally result from the consumption of the flesh of animals affected with septicæmia or pyæmia (uterine, umbilical, udder or bowel affections). Bollinger has proved that the so-called mycosis of the bowels is not anthrax, but a septic inflammation of the intestinal mucous membrane. The bacteriological investigations of the last decade point to a long list of bacteria as the causes of meat poisoning.

1. Gärtner observed, during an epidemic of poisoning in Frankenhausen, characteristic bacteria both in the poisonous meat and in the organs of a man who died from eating it, to which he gave the name of "bacillus enteritidis." These bacteria showed lively rotary movement in the suspended drop, took aniline colour very strongly at one end, while the rest seemed hardly coloured at all. Moreover, the meat contained poisonous matter which resisted boiling, and produced in animals inoculated therewith enteritis, convulsions and the symptoms of paralysis. Dogs and cats were immune to the bacteria, while rabbits, guinea-pigs and mice died with all the appearance of violent enteritis. Johne also met with the bacillus enteritidis in a case of meat poisoning at Bischofswerda.

2. Gaffky and Daak discovered in a wholesale poisoning case at Röhrsdorf, caused by eating horseflesh sausages, characteristic "sausage bacilli," in the form of movable rod-shaped bacilli, with rounded ends, which sometimes developed into threads, and flourished best in slightly alkaline broth. Given to monkeys, guinea-pigs and mice, these sausage bacilli produced enteritis and diarrhœa. Dogs, cats, pigs and rabbits, on the other hand seemed to be immune.

3. In a case of meat poisoning in Rotterdam, Poels and Dhont found the so-called " Rotterdam-bacilli.' These are short and very fine rods, which in a large number of experiments, proved fatal to cattle within fourteen hours.

4. Van Ermenger found in a meat poisoning case at Moorseele in two calves the so-called Moorseele bacilli ; these were very active and had numerous thread-like tails. They produced a toxic albumen which a temperature of 130° C. (266° F.), failed to destroy, and caused enteritis in the animals experimented on in whatever way they were absorbed by the animals.

5. Flügge found in the Breslau meat poisoning case the Breslauer bacillus which bore some resemblance to the *bacillus coli* and the Moorseele and Rotterdam bacilli.

6. Basenau gives the name of *bacillus bovis morbificans* to a bacillus bred in cows' flesh. He is of opinion that all the bacteria which have been discovered up to now in connection with meat poisoning, bear a strong resemblance to the *bacillus coli.*

7. Kuborn shows that *staphylococcus pyogenes albus* was present in a case of meat poisoning and the cause of it.

Bail has pointed out that the bacteria of meat poisoning may be carried by flies. Glage finds that symptoms of meat poisoning are not developed in swine which have been fed on meat-flour (prepared according to Podewil's system) from putrid or infected meat.

Etiology of Sausage-Poisoning.—Sausage-poisoning (*botulismus, allantiasis*) occurs less frequently than meat-poisoning in man. It is caused, not only by decomposing sausages, but by putrid meat, decomposing ham, liver, brawn, tinned meat and goose-flesh. According to experiments recently undertaken by van Ermeugen, sausage-poisoning is not set up by the toxic products of the decomposition of albumen, but is originated by a specific bacillus—the *bacillus botulinus.* This bacterium is anaerobic. It is a straight rod with slightly rounded ends and formation of spores, and produces a virulent toxin, fatal to rabbits in doses of 1-2000 mg. It develops in pork whether sweetened, rendered akaline or boiled. Cultures of the bacillus smell strongly of butyric acid (not the smell of decomposition). After being kept for one hour at a temperature of 70° C. (158° F.), botulismus toxin becomes inactive. Pfuhl found that extensive poisoning by " beef-sausage " in Hanover in 1900 was caused by the presence of *proteus mirabilis.*

4.—MYCOTIC GASTRO-ENTERITIS OF HERBIVORA, AFTER CONSUMPTION OF FOOD CONTAINING FUNGI (MOULD FUNGI, URIDINEÆ, USTILAGINEÆ). SO-CALLED POISONING BY FUNGI.

Etiology.—Under the names of enteric fever, typhus, gastro-enteritis, etc., we find among herbivorous animals a number of cases of disease described, which, on closer inspection, appear to be nearly allied to meat-poisoning in dogs. To these belong the remarkable and, at first, almost inexplicable ailments which were noticed in horses, cattle, sheep and pigs after eating

damaged or rotten fodder (straw, hay, oats, oil-cake, chaff, turnips, potatoes, meal, bread). For instance, the so-called "shavings sickness" of cattle, which in North Germany followed in many districts from feeding them on putrid beetroot shavings from the sugar factories. Further, in cows and horses illness was caused by giving spoilt and fermenting potatoes. There is no doubt that these are cases of the operation of infectious mycotic materials upon the intestinal mucous membrane, and of absorption into the blood of ptomaïne-like bodies. In our literature numerous instances are given of the injurious effect upon our domestic animals of the fungi which vegetate upon their herbivorous food, and the above-named cases of injury from bad, rotten or fermenting food can be attributed to the presence therein of fungi. The following species of fungus growth are to be considered :—

1. *Mould-fungi* (*mucorinæ*), and especially mucor, aspergillus, and penicillium, which, though in themselves not injurious, yet by inducing decomposition of food materials (bread, flour, oats, hay, straw) give rise to ptomaïnes. In cultures of mould-fungi, Seber discovered inflammatory toxins of the same composition and activity as the phlogosin of the micrococci contained in pus, to which the inflammation accompanying infection by pus-micrococci is due.

2. *Uridineæ*, of which puccinia graminis is the most important. The experiments made by Franck with uridineæ on rabbits produced precisely the symptoms of meat-poisoning, viz.: gastric disturbances, with staggering and spasms.

3. *Ustilagineæ*. The chief representatives of this group are the greasy smut of wheat (tilletia caries), which is clinically by far the most important, and also ustilago carbo (dusty or flying smut) and ustilago maydis (the smut of maize). The smut fungi (*ustilagineæ*) have the most intense effect of all fungi upon the digestive mucous membrane and general organism. This powerful effect must be attributed to some irritant ptomaïnes which develop in the plant as waste products of the fungi.

4. *Germ-fungi* (*pyrenomycetes*). Clinically important is Polydesmus exitiosus, the "rape destroyer," which feeds on the green parts of rape.

5. *The yeast-fungus*, the cause of alcoholic fermentation. This is probably the last stage of development of the mould-fungus.

The etiology of mycotic gastro-enteritis is very obscure.

An epidemic of sickness will sometimes appear among domestic animals which, both in its course and in the nature of the anatomical changes to which it gives rise, presents an exact clinical counterpart of fungus-poisoning. At the same time it is frequently impossible to discover the fungus in question, or, at any rate, in a quantity sufficient to account for the occurrence of the sickness. There is much here which calls for scientific investigation. It should be remembered in connection with poisoning by *tilletia caries* and *puscinia graminis*, that the spores of these growths are frequently present in fodder which, in itself, is quite harmless (c.p. Pusch's experiments). It is not improbable that these cases of so-called fungus poisoning in herbivora are really infectious diseases, the nature and origin of which are at present unknown (c.p. accounts published in recent years by Schmidt, Grözinger, Louis, Munkenbeck, Albrecht, Siecheneder, Wilhelm, Bayne, Gotteswinter, etc.).

The inflammatory conditions of the gastro-intestinal tract, which were formerly known as "typhoid," have nothing in common with human typhus. Many of them should undoubtedly be classed as fungus-poisoning. Thus Gerlach describes a "typhoid" inflammation of the gastro-intestinal mucous membrane in horses which was accompanied by symptoms of colic, constipation, a high and very feeble pulse, grinding of the teeth, etc. The condition differed essentially from common colic, as well as from the gastric-bilious form of influenza and from anthrax. Among the causes he mentions the feeding with unripe, insufficiently dried oats, and with ill-conditioned mouldy straw in place of hay. Derache also gives an account of a similar non-infectious sickness among horses, which was described as "typhus"; its symptoms, however, had nothing in common with either petechial fever or anthrax. The disease, as a rule, would attack several horses in a stable at the same time; but, with the exception of the fodder which was not only insufficient in quantity, but consisted of dusty, damp, ill-smelling hay, and oats of the poorest quality, it was impossible to discover any etiological factor whatsoever. The symptoms were those of mycotic gastro-enteritis: sudden attack, weakness resembling paralysis; extremely feeble pulse; constipation with diarrhœa; passage of slimy masses of fæces; rapid course and fatal termination. The *post-mortem* appearances were very characteristic, closely resembling those of human typhus. There was marked swelling of the solitary glands and Peyer's patches, with purulent infiltration; formation of ulcers and scabs lead-

ing to perforation; extreme ecchymosis; tree-like injection of the vessels together with grey and brownish discolouration; liquid, sticky contents of the bowels, mixed with pus. There was very rapid decomposition of the cadaver; the flesh became soft and discoloured; the liver, putrid; the heart, as if boiled; the blood stagnant and containing bacteria. Attempted infection, even by intravenous inoculation, was without result. There was no splenic enlargement, and carbuncular swellings of the sub-cutis and the intestinal mucous membrane were absent.

Although this description bears a strong resemblance to that of human typhus, it cannot be identified with it. The evidence is inconclusive and, up to the present, all attempts to infect animals (cattle, swine, calves), with human typhus, as undertaken by Bollinger, Obermeier, Lebert, and others, have been unsuccessful. Moreover, a true clinical case of human typhus in the domestic animals has, up to now, never been observed.

Symptoms.—The aspect of poisoning by fungi is very varied, and its appearances in general as follows:—Illness appears suddenly and generally in several animals at once, which resembles an epidemic. The animals forsake their food and drink, become restless, get abdominal pains, which may be shown by moans and even bellowing. Peristaltic action is suppressed, and there is at first obstinate constipation, followed later by profuse diarrhœa, with discharge of fœtid, bloody and slimy matter. Slight tympany is sometimes present. Occasionally the muscles of the tongue and pharynx are paralysed; causing a free discharge of saliva, continuous chewing and cough. These gastric phenomena are accompanied by high fever, with an abnormally quickened pulse (in cattle 100 beats and above), ague fits, palpitation, coldness of the extremities, etc. At other times there is extreme weakness and prostration, with general muscular paralysis (as though from hemlock poisoning), which is the most important symptom to note. The animals lie motionless on the ground in a paralysed condition. Sometimes cerebral symptoms are observed, such as dizziness, insensibility, behaviour suggestive of chronic shivering, amaurosis and cerebral spasms. The chief symptom is frequently nephritis and cystitis (polyuria), and sometimes abortion. Many cases of fungus-poisoning are at last marked by tympanites, palpitation, dyspnœa, and others by inflammatory swellings of the skin of the head (mucous membrane of lips, mouth and nose). The

duration of the disease is often short (about a day); but sometimes lasts eight, or even fourteen days. Death frequently ensues with the appearance of apoplexy.

The great variety of these phenomena may perhaps be best explained by the fact that the metabolic products of mould-fungi vary considerably, both chemically and physiologically, according to the changing outward conditions (temperature, place of growth, degree of development, etc.) of the fungus. It is possible that the simultaneous drinking of warm water (the water of brewers' grains) may have a peculiar influence upon the development of the fungi, and therefore upon the aspect of the disease. This is in accordance with the fact that mould-fungi do not, under all circumstances and in all animals, produce any pathogenic effect. There may be some predisposing cause present in the digestive organs. As, for instance, in thrush (*aphthæ*) all the animals in a stable are not affected, so it would appear that some are immune against mould-fungi. On this account experiments in feeding with this fungus have often given only a negative result. But to deny the pathogenic nature of mould-fungi on these grounds would be as unjustifiable as it is illogical.

The aspect of individual cases of mycotic poisoning varies much, and is often very characteristic. Sometimes if several sorts of fungi are associated, the symptoms are irregular. Those which are peculiar to the various forms of fungoid poisoning are as follows :—

1. *Mould-fungi.*—Loss of appetite, colic, tympanites, constipation, diarrhœa, blood-stained, slimy and sometimes very malodorous fæces, polyuria, dizziness, insensibility, conduct as in chronic hydrocephalus, paralysis of limbs, tongue, etc., and amaurosis. Also profuse sweating.

2. *Smut-fungi* (*Ustilagineæ*).—Flow of saliva, persistent chewing, staggering, reeling and general paralysis of locomotion and sensation. In other cases, gastric symptoms.

3. *Rust or mildew-fungi* (*Uridineæ*).—Inflammation of the skin of the head (lips, cheeks, eyelids), conjunctivitis, urticaria, stomatitis, pharyngitis, glossitis, colic, bloody diarrhœa, hæmaturia, paralysis and somnolence.

4. *Germ-fungi* (*Pyrenomycetes*).—Similar appearances; stomatitis, pharyngitis, rhinitis, dermatitis on the head, gastro-enteritis, hæmaturia, paralysis of the bladder and of the hind-quarters.

5. *Yeast-fungi.*—Considerable cerebral excitement with subsequent stupefaction and signs of paralysis.

For further details on the various fungoid poisons, see Fröhner's "Text-Book of Toxicology for Veterinary Surgeons."

Post-Mortem Notes.—As with the external symptoms, so also there is considerable variation in the *post-mortem* lesions.

As a rule, one may expect the signs of severe hæmorrhagic gastro-enteritis, with general septic changes (swelling of the liver and spleen, fatty degeneration of the muscles of the heart), but often the result is purely negative, as in narcotic poisoning. The following is to be noted concerning the various forms of fungoid poisoning. With *mould-fungus* we find inflammation, ecchymosis and erosion of the gastro-intestinal mucous membrane, as well as hyperæmia and œdematous infiltration of the brain and spinal cord. In poisoning by *ustilagineæ* a *post-mortem* examination yields generally negative or very slight results. Sometimes hæmorrhagic gastro-enteritis is found, as well as streaks of sooty discoloration on the mucous membrane of the smaller intestine (so-called eelskin). Dissection in cases of poisoning by *uridineæ* and *pyrenomycetes* reveals dermatitis, stomatitis, pharyngitis, hæmorrhagic gastro-enteritis, nephritis and cystitis. In consequence of the frequent association of several fungi, the *post-mortem* results are far from being uniform.

Differential Diagnosis.—Mycotic inflammation of the bowels is frequently confounded with other complaints owing to its peculiar septic-infectious character. The following must be noted as likely to be mistaken for it :—

1. *Various toxic gastro-enterites*, with similar rapid course and the same grave general affection. Differentiation is only possible by discovery of the cause.

2. *Rinderpest*, especially when several animals are simultaneously attacked with intestinal mycosis from eating the same damaged fodder. But certain symptoms peculiar to rinderpest will be absent, viz. : widespread inflammation of all the mucous membranes (not only those of the digestive organs, but also of the breathing apparatus, eyes and vagina), the characteristic erosions on the membrane of the mouth and vagina, its terrible contagiousness and the *post-mortem* results.

3. *Anthrax*. Diagnosis is based upon the course of the disease, either apoplectic or epileptiform ; upon its extremely varied and complex symptom ; upon its appearance in certain districts only ; and upon bacteriological proof of the presence of the specific *anthrax bacillus*.

4. *Foot-and-mouth disease*. Poisonings caused by *uridineæ* and *pyrenomycetes* (*Stomatitis ulcerosa*) are particularly apt to be mistaken for this disease. They were formerly designated

sporadic thrush (*Aphthæ*), but are easily distinguished from foot-and-mouth disease by their non-contagious character.

5. *Pox.* Poisonings by the two last-named fungi may in sheep be confused with pox, owing to the inflammatory skin affection of the head which they produce. But the lack of contagiousness, as well as the absence, for the time being, of sheep-pox in the country help us to a sure diagnosis.

6. *Dysentery.* This infectious form of gastro-enteritis is accompanied from the beginning by diarrhœa, generally lasts longer and has rather the character of contagious epidemic disease.

7. *Subacute inflammation of the brain* in horses and *cerebro-spinal meningitis* in cattle. In most cases bowel symptoms are absent. Yet in many instances of inflammation of the brain in horses dissection has revealed septic changes of the internal organs, as well as gastric disorder, suggesting resemblances to mycosis or infectious disease.

8. *Rabies.* The differential diagnosis—often not otherwise easy—depends, in the first place, upon the history, and next upon the prevalence of bowel symptoms at the commencement of the mycotic gastro-enteritis.

9. *Influenza.* This is distinguishable from the form of enteritis just named by its contagiousness, and its quite specific symptoms, particularly the affection of the eyes. But in some cases differentiation is not easy.

Treatment.—With regard to the treatment of mycotic enteritis in different animals, we recommend emetics for dogs at the beginning, and later on purgatives, especially calomel. The latter is also good for horses, owing to its disinfectant qualities. For ruminants saline remedies are preferable. In dogs a washing out of the bowels with warm solutions (1 per 1,000) of tannic acid, or a weak solution of creolin, is useful. Very often, however, the best result is obtained with stimulants (wine, spirits of wine, ether, veratrine, atropine, caffeïne or camphor). General treatment is the same as that already given for gastro-enteritis.

5.—TOXIC GASTRO-ENTERITIS (POISONING).

General Remarks.—Toxic gastro-enteritis may be caused by numerous poisons, which may be divided into two great groups:

1. Irritant poisons and
2. Narcoto irritant poisons. A knowledge of the effects of the different specific poisons is of the greatest value to the veterinary surgeon. For this reason we have thought it advisable in place of a descriptive summary of general toxic gastro-intestinal inflammation, to give a short practical account of the clinical, therapeutic and anatomical aspects of poisoning by each of the irritant and narcoto-irritant poisons. More elaborate details are to be found in the text-books of the subject.

First of all, a few remarks on the diagnosis of such cases are indispensable. It is not always easy to recognise a case of poisoning, as the symptoms in life and after death often bear great resemblance to those of infectious diseases, as well as to non-toxic gastro-enteritis. In general the following points are important aids to diagnosis :—

1. *The previous history of the case or patient, which is frequently very scanty.*
2. *Suddenness of attack, rapid course and often sudden death.*
3. *Connection between the symptoms and the food taken.*
4. *Simultaneous sickness of many animals without demonstrable contagion.*
5. *Complication of gastric and nervous symptoms.*
6. *The characteristic symptoms of certain poisonings and the pathological lesions.*
7. *Physical, chemical and pathological proof of the presence of the poison.*

I.—MINERAL POISONS.

POISONING BY PHOSPHORUS.

Symptoms.—Attacks of colic, vomiting, groaning, icterus, albuminuria, sometimes inability to swallow, with swelling of the tongue ; paralytic appearances, weakness of the heart, sub-normal body temperature ; trembling ; accelerated pulse and breathing ; and in prolonged cases hæmorrhagic diathesis. After inhaling phosphorus vapour : cough, attacks of choking, emphysema in the lungs, skin and on neck and thorax. In *Birds :* faintness, prostration, thirst, diarrhœa,

and at times remarkable jumping movements. Duration : from a few hours to several days. If the sickness be prolonged (say three to five days), a remission of the severer signs often takes place, with apparent improvement. Schindelka noticed with a number of milch-cows that after complete recovery there was an *entire cessation of milk*, so that they had to be slaughtered.

Post-Mortem Appearances.—Very similar to those of arsenical poisoning, viz : gastro-enteritis (which may, however, be absent), in certain cases even stomatitis and pharyngitis ; glandular gastritis (parenchymatous degeneration of the glands of the stomach) ; if the affection last many days, fatty degeneration of the internal organs supervenes, particularly of the liver, heart, epithelium of the kidneys and muscles ; bronchitis on breathing phosphorus vapour ; ecchymosis and hæmorrhagic foci in the various organs, dark, fluid blood, phosphorescence of contents of stomach and bowels with emission of vapour and a characteristic smell of phosphorus.

Therapeutics.—Administration of emetics, especially sulphate of copper (to form insoluble compound with the phosphorus), permanganate of potash, in 1–5,000 solution, peroxide of hydrogen in 1–3 per cent. solution, nitrate of cobalt, old spirits of turpentine rich in oxygen (given with large quantities of mucilage) to oxydise the phosphorus into non-poisonous phosphoric acid. As a dose for horses give 1½–3 ounces of the spirit of turpentine ; for cattle 3–6 ounces ; for pigs 6–12 drams ; for dogs 1–2½ drams ; and for fowls 5–10 drops ; also stimulants. Oils must be carefully withheld as they would only dissolve the phosphorus and render it more active ; the same remark applies also to milk.

ARSENICAL POISONING.

(A)—ACUTE FORM.

Symptoms. — These are primarily of gastro-enteritic nature ; vomiting, salivation, colic, constipation ; in cattle, tympanites and later offensive diarrhœa, sometimes even bloody ; in ruminants, occasional painful swelling of the inferior abdominal region with formation of abscesses and of a fistula of the abomasum, or even prolapse (fistula very rarely

occurs in the second stomach). These symptoms are complicated by disturbance of the nervous system: paralysis (tabes arsenicalis), tottering, trembling, weakness, stupor, enlarged pupils, etc. The pulse is feeble or imperceptible and very rapid (weakness of the heart); the bodily temperature unevenly distributed; the breathing often very much accelerated and strained. Duration: a few hours to several days. On external application, as *e.g.*, from wounds, great swelling is noted, and on bathing, the skin is inflamed in various degrees —this in addition to the above-mentioned symptoms.

Post-Mortem Appearances.—Dissection reveals corrosive gastro-enteritis, with redness, swelling, ecchymosis and erosion of mucous membranes, especially those of the stomach, *i.e.*, of the abomasum. Occasionally perforation of the walls of abomasum and abdomen. Fatty degeneration of the gastric glands (glandular gastritis, adenitis parenchymatosa). Fatty degenerations of internal organs, such as the liver, kidneys, heart and brain. Ecchymosis, especially under the endocardium. Mummification of the carcase.

Therapeutics.—The principal antidote is the hydrated peroxide of iron (liquor of sulphate of iron, 100 parts mixed with 250 parts of water, to which is added 15 parts of calcined magnesia, in 250 parts of water). The hydrated oxide of iron so formed makes an insoluble compound with the arsenic. To dogs give of this arsenical antidote a table-spoonful every 15 minutes; to pigs and smaller ruminants several spoonfuls; to horses and cattle 10–40 ounces. A simple solution of calcined magnesia in 20 parts of water also produces an insoluble compound with the arsenic (dose of the calcined magnesia for dogs 7 to 15 grains every 15 minutes; for horses 1 to 3 drams; and for cattle 2 to 6 drams). Other antidotes are sulphur, sulphate of potash and sulphate of iron. If these remedies are not at hand, use any preparation of iron, such as the familiar household specific, water in which red-hot iron has been cooled, or lime-water, sugar-water, the white of eggs and mucilaginous remedies. Oil must be carefully avoided.

(B.)—CHRONIC ARSENICAL POISONING IN CATTLE CAUSED BY BREATHING ARSENICAL FUMES.

Symptoms.—Chronic diarrhœa and cough. A hidebound condition of the skin, with epidermal desquamation (chronic eczema squamosum), muscular atrophy beginning at the hind quarters, paralytic weakness, also starting from behind, increasing loss of flesh, and cachexia. Cessation of milk, abortion, delay in passing the after-birth, difficulty in conception.

Post-Mortem Appearances.—Great emaciation of the carcase, general hydræmia; sloughs, ulcers and scars in the abomasum, less frequently in the first stomach; red spots, inflammation of mucous membranes in abomasum and small intestine; ulcers on the membrane of trachea. Tubercular, caseous broncho-pneumonia occurs as a secondary lesion.

Therapeutics.—Prophylaxis, *i.e.*, cease feeding with fodder covered with the deposited chemical dust; treat diarrhœa, cough, loss of flesh, weakness, &c., according to symptoms.

The so-called "chemical-fumes sickness" (Hüttenrauchkrankheit) in the neighbourhood of the Freiberg works must, according to Haubner's description, be regarded as essentially a chronic arsenical poisoning. The fumes consist mainly of sulphurous and arsenical acids, with smaller quantities of lead and zinc vapours and traces of earthy matter. The lead and zinc cannot, as Haubner proved, be regarded as the poisonous agents. Sulphurous acid is not present in the fine dust which settles upon and covers all the neighbouring vegetation, and must be regarded as the exciting cause of the sickness in question, which consequently must be arsenical poisoning, since arsenic is found in the organs of all animals affected. The so-called "caseous pneumonia caused by fumes from the works," which Haubner and Siedamgrotzky classified as a peculiar inhalation-pneumonia, has been shown by Johne's investigations to be genuine tuberculous broncho-pneumonia. Nevertheless, the inhalation of the fine dust by the animals when eating the fodder on which it has fallen is of great importance as a pre-disposing cause, which facilitates infection by the bacilli of tuberculosis. The consumption of this dust, when the amount of arsenic is larger, may even lead to acute arsenical poisoning (see previous paragraph). The so-called "sour or acid-sickness," which Haubner attributed to the sulphur in the fumes (and not to the dust), and which took the form of anæmia, with brittleness of the bones, has nothing in common with the above-named disease caused by eating fodder covered with arsenic-containing dust (chronic arsenical poisoning).

LEAD-POISONING (*Saturnism, Plumbism*).

1. **Symptoms of Acute Lead Poisoning** are at first those of gastro-enteritis: vomiting, salivation, restlessness, bowel-pains, continuous constipation; in ruminants, tympanites, later (at times), stinking diarrhœa and suppression of the secretions (urine, milk). To these supervene nervous disorders, convulsive movements, especially of head and neck, trembling (tremor saturninus), spasms of the jaw, attacks resembling epilepsy, intermittent irritability, even mania (mania saturnina), convulsions and spasms like those of chorea. Then also there appear weakness, somnolence, partial or complete paralysis, particularly of the hind-legs (paralysis saturnina), and insensibility to pain (anæsthesia saturnina). The pulse is hard and thready, often slow, and the breathing difficult and even dyspnœic.

2. **Symptoms of Chronic Lead Poisoning.**—Increasing emaciation and weakness (tabes and cachexia saturnina), constipation, with colic, affected locomotion (arthralgia saturnina and rheumatismus saturnina), brain disorders as in the acute form (encephalopathia saturnina), especially epileptic fits (eclampsia saturnina); in horses, whistling or roaring; in goats, abortion and sterility. Moreover, there is development of ulcerous stomatitis (blue line on the gums), muscular atrophy and permanent muscular contraction (atrophia and contractura saturnina), and of chronic nephritis (contracted kidney). In pigeons, single phalanges of the toes sometimes drop off.

Post-Mortem Appearances.—Paleness and contraction of the intestinal canal; in chronic cases, thickening of the mucous membrane of the digestive apparatus, localised redness, inflammation, formation of scabs and of ulcers, with metallic discoloration of the mucous membrane from lead-grey to deep black. Hydrocephalus and hydrorrhachis, internal and external; contraction of the kidneys. In cases of death resembling apoplexy no essential changes are found.

Therapeutics.—Administration of sulphuric acid in mucilage or in water, as well as sulphates of sodium, potassium and magnesium, in order to produce insoluble sulphate of lead. Emetics and purgatives. Albumen, milk, mucilage. Intern-

ally, iodide of potassium (which hastens the removal of the lead, as iodide of lead). Administer symptomatically—morphia, atropine, nitrite of amyl, chloral hydrate, etc., to prevent lead colic, eclampsia, etc. In horses resort to tracheotomy.

MERCURIAL POISONING (*Mercurialism*).

Symptoms.—The phenomena of genuine mercurial poisoning are as follows, apart from the local corrosive action of the several preparations :

1. Salivation, ulcerous stomatitis, loosening of the flesh of the gums, loosening and even loss of the teeth in ruminants, bad-smelling mouth.

2. Gastro-intestinal catarrh ; later, profuse and even bloody diarrhœa, with greyish green, liquid, fœtid fæces, smelling sometimes like putrid flesh.

3. Cough, putrescent nasal discharge, broncho-blennorrhœa and even broncho-pneumonia, strained and even dyspnœic breathing, badly-smelling breath, groaning and bleeding at the nose.

4. Eczema of the skin, chiefly impetiginous and squamous, beginning with excessive itching, which leads to gnawing and rubbing, loss of hair, weeping, formation of pus and scabs, together with considerable thickening of the skin and swelling of the subcutis ; also growth of vesicles and pustules, the latter, however, without a depression. Most frequent location is where the ointment has been rubbed in, round the eyes, the mouth, the anus, the udder, the vagina and the creases about the joints.

5. Weakness resembling paralysis, great apathy, stupor, dizziness, trembling (tremor mercurialis), timidity and delirium (erithismus mercurialis). Also paralysis of single muscles, amaurosis, deafness, anæsthesia and emaciation. Death often occurs in coma, and rarely in convulsions.

6. Aneurism as the result of nephritis and incrustation of lime in the kidneys, often lasting several days, cylindruria, albuminuria.

7. Temperature normal, or occasionally raised, but the pulse, on the contrary, much accelerated and feeble, hæmorrhage in different organs, especially in the mucous membranes of the nose, lungs, bowels, and uterus (abortion). Death follows sometimes from internal hæmorrhage.

Course of the Disease.—This is very rapid in young animals when there is great local corrosion of stomach and bowels (sublimate may produce fatal results in an hour, without the stage of general mercurialism being reached). The duration of the acute form varies from 10 to 14 days. It may, however, be chronic, lasting several weeks, or even months. Generally, the symptomatic appearances are not so complete as described above, one or other of the symptoms being as a rule absent.

Post-Mortem Appearances.—The skin and subcutaneous tissue is found to be anæmic, the latter infiltrated with serum and blood-stained fluid under the eczematous parts of the skin. The muscular apparatus is exceedingly pale, flabby as if boiled, permeated with spotted, dark-brownish-red ecchymoses, and at the same time largely infiltrated with a gelatinous fluid. The blood appears dark-red, greasy and badly coagulated. Inflammatory signs are present in different degrees upon the mucous membrane of the digestive tract, such as ulcerous stomatitis (this may also be absent), dots and spots of red, hæmorrhagic erosions, ulcers in the stomach, especially on the top of the prominences of the mucous membrane and in the abomasum; the mucosa is often œdematous and swollen, the submucosa infiltrated with serum, so that the mucous membrane of the bowels forms loose folds. Excessive intestinal anæmia is also observed. The sub-peritoneal connective tissue is likewise œdematous and infiltrated with spotted hæmorrhages. The liver is anæmic and swollen; the kidneys are infiltrated with serum, anæmic, and ecchymosed; sometimes they are completely incrusted with lime; there is fatty degeneration of the renal epithelium. In addition there are also sometimes rhinitis, laryngitis, tracheitis, catarrhal bronchitis and even croup of the larynx and trachea. The lungs are full of blood, infiltrated with hæmorrhagic and broncho-pneumonic foci, or even with abscesses, the bronchial and mediastinal glands are swollen and hyperæmic; subpleural ecchymosis is also present. Finally, myocarditic degeneration and hæmorrhage are found between the muscular fibres of the heart, beneath the epicardium and endocardium; subarachnoideal infiltration of blood, with anæmic, soft brain-substance, gleaming like water, and dotted hæmorrhages on the superficial layer of the brain (cortex).

Therapeutics of General Mercurial Poisoning.—Administration of sulphur compounds, so as to form an

insoluble compound of the mercury and sulphur, is recommended, viz., flowers of sulphur, potassium sulphate, etc. Also iron filings, and sulphate of iron; albumen in solution; and iodide of potassium. No common salt. In addition to these, adopt symptomatic treatment for gastro-enteritis, stomatitis, bronchitis, eczema, etc.

POISONING BY COPPER (*Cuprism*).

Symptoms.—They are those of gastro-enteritis, viz., vomiting greenish matter, loss of appetite, colic and diarrhœa. Later follow great weakness, trembling, convulsions, anæsthesia; small, thready, slow pulse, quickened breathing.—*Autopsy* reveals the signs of acute gastro-enteritis. According to Trasbôt, considerable enlargement of the stomach is observed. —*Therapeutics*: albumen, mucilage, milk, reduced iron, iron filings, sulphur, calcined magnesia, ferrocyanide of potash. According to symptoms administer narcotics.

Chronic Copper Poisoning.—This has been experimentally induced in animals by Baum and Seeliger by means of continued minute doses of copper. Cats are peculiarly receptive. The symptoms are: debility, loss of appetite, falling of the hair, and convulsions. The *post-mortem* results are: chronic catarrh of the small intestine; extreme degeneration of the liver and kidneys. According to von Kossa, calcification of the kidneys and liver is a characteristic symptom of chronic copper-poisoning in rabbits.

POISONING BY ZINC.

Symptoms.—Vomiting, colic, diarrhœa, weakness and signs of paralysis; if long continued, anæmia and cachexia. —*Post-mortem* shows shrinking and anæmia of the gastro-intestinal mucous membrane, with gastro-enteritis in the form of localised inflammatory foci and erosions.—*Therapeutics*: albumen, mucilage, sugar-water, sulphur and calcined magnesia.

POISONING BY TARTAR EMETIC.

Symptoms.—Similar to those of poisoning by corrosive alkalies and acids: ulcerous stomatitis with salivation, vomiting (in isolated cases also in horses), colic, diarrhœa, dizziness,

trembling, spasms, paralysis, small, hard and often imperceptible pulse, accelerated breathing, even pneumonia; rapid death.—*Autopsy* shows, inflammation and erosion of the mucous membranes of the entire digestive tract, especially the stomach (pharyngitis, stomatitis, œsphagitis, gastritis and ulcerous enteritis); also hyperæmia of the lungs, scaling or peeling of the bronchial epithelium, hæmorrhagic congestion, and even inflammation of the lungs. According to Trasbôt, enlargement of the stomach is also a characteristic symptom of this form of poisoning.—*Therapeutics:* first of all, remedies containing tannin (decoction of oak-bark, ink, tannin); mucilaginous and oily substances; narcotics, especially opium; with dogs, small pieces of ice.

Sulphur Poisoning.—Gastro-enteritis with violent colic and the passage of watery, blackish stools, smelling of sulphuretted hydrogen; coma, whilst the breath will smell of sulphuretted hydrogen. The same symptoms are observed in poisoning with potassium sulphuret. Treatment: iron, magnesia, stimulants, the mucilages (Demblon, Mosselmann, Hébrant, Fabretti).

POISONING BY COMMON SALT, BRINE, AND THE BRINE OF HERRINGS AND MEAT.

Symptoms.—In cases of poisoning by *common salt,* we have chiefly signs of gastro-enteritis, combined with conditions resembling paralysis. Cessation of eating, a dry, hot, red mouth, great thirst, vomiting, colic pains, watery diarrhœa, and frequent urination, are the signs first seen. Then come great bodily weakness, staggering, falling, paralysis of varying distribution (particularly of the hind limbs in horses), stupor, dilation of the pupils, even blindness, and tremblings; in pigs (but less frequently in horses and dogs), now and then fits of cramp resembling epilepsy; weakness of the heart, small, weak pulse, dyspnœa. Death occurs within from six to forty-eight hours. Should the sickness last longer, a condition of anæmia sets in. In cattle, the appearances of enteritis sometimes manifest themselves.

2. In *poisoning by herring brine* the symptoms of ptomaine poisoning are added to the above (brain irritation and spasms). Especially characteristic signs are: grinding the teeth, spasms of the jaw and cramps of epileptic nature, opisthotonus and

pleurosthotonus, rolling the eyes (nystagmus), spasmodic blinking, widespread twitching, twisting the body round, squatting on the haunches, phenomena of chronic hydrocephalus, stupor, insensibility, amaurotic pupils and blindness. In cows, abortion, even prolapse of the uterus. Course very acute, death generally occurring after from six to twelve hours.

Post-Mortem Appearances are those of acute gastro-enteritis, the abomasum in ruminants being especially affected. Also conspicuously bright-red blood, very fluid, with ecchymoses in various organs. The membrane of the bladder is often reddened. Inflammatory changes are often absent in the intestinal canal, especially in brine poisoning; on the other hand, we generally find in the latter, hyperæmia of the brain with internal and external hydrocephalus.

Therapeutics.—Plentiful supply of water, mucilaginous and oily remedies, stimulants (ether subcutaneously, camphor, wine, etc.). Where the excitement is great narcotics may be given.

POISONING WITH SALTPETRE (NITRATE OF SODA AND NITRATE OF POTASH).

Symptoms are those of gastro-enteritis combined with paralysis; colic, diarrhœa, tympanites (in cattle), vomiting, slavering and choking movements; also fall of bodily temperature, prostration, tottering, falling, stupor, tremblings, palpitating heart-beat and polyuria. Death occasionally takes place from apoplexy and more rarely is accompanied by spasms, rolling of the eyes, etc. The course is commonly acute, the animal often dying within five minutes. Duration: mostly from half an hour to several hours, rarely more than twelve. *Autopsy* reveals: hæmorrhagic gastro-enteritis, especially in the stomach (abomasum) and small intestine; purple to cherry-brown red discolouration and ulcerous degeneration of the mucous membrane; contents of stomach, a brownish-red fluid; hyperæmia of the abdominal viscera, inflammation and ecchymoses of the kidneys and mucous membrane of the urinary bladder; blood fluid and bright red or discoloured.—*Therapeutics:* emollients, narcotics or stimulants (camphor, coffee, ether, etc.).

POISONING BY GLAUBER SALTS, EPSOM SALTS AND SALTS USED AS MANURES.

Symptoms.—Colic, intense thirst, watery diarrhœa, suppressed peristalsis, paralytic condition, inability to stand up, increasing debility; death after several days. Similar appearances are noted to those of poisoning by kainite and superphosphates.—*Treatment:* as in cases of poisoning by common salt.

Chloride of Barium Poisoning.—The symptoms of poisoning by chloride of barium vary according to the mode of its introduction: whether through the veins, subcutaneously, or through the mouth.

When it is injected into the veins in excessive doses, horses generally die suddenly, even in the course of a few minutes, staggering, completely collapsing, or from convulsions and general paralysis (death from apoplexy). In subcutaneous inoculation the specific convulsive action caused by barium in the *involuntary muscles of the intestines*, the effect on the heart—paralytic as in the case of digitalis—can be more closely followed. The spasm of the *large gut* takes the form of severe and painful colic, with violent diarrhœa and the discharge of large quantities of watery excreta, when biting, salivation, choking, vomiting and moaning are observed. The irritation of the cramp-centres is followed by spasms—as caused by strychnine, which at first chiefly attack the muscles of the back and extremities; but soon change into creeping cramp and general paralysis (giddiness, weakness of the loins, staggers and insensibility). Finally, paralysis of the heart manifests itself by excessive pulsation and dyspnœa.

Post-Mortem Conditions.—The intestinal canal is found empty, as if rinsed out, the mucous membrane sometimes swollen and inflamed, and the heart parenchymatous, as if it had been steeped in boiling water. In *periacute* cases (vein-injection) the result of the examination is purely negative.

Therapeutics.—As in the case of poisoning by strychnine in acute and *sub-acute* forms, remedies to relieve the convulsions are indicated: morphia, opium, bromide of potassium, chloroform, chloral-hydrate. In *acute* cases these are, however, useless.

POISONING BY CORROSIVE ALKALIES (CAUSTIC POTASH, CAUSTIC SODA, SPIRIT OF AMMONIA).

Symptoms.—Erosion of the lips and mucous membrane of mouth, tongue and pharynx ; foaming, difficulty of swallowing, colic. When ammonia is the poison taken, coughing also, difficult breathing, and the coughing-up of skinny, croup-like matter. Muscular weakness and collapse. *Autopsy* reveals : croupy stomatitis and pharyngitis. In ammonia poisoning also croupy laryngitis, tracheitis, bronchitis and pneumonia ; intense inflammation, brown to reddish-black discolouration, gelatinous swelling and extensive corrosion of the stomach and intestinal membranes.—*Therapeutics :* diluted acids, especially (as household remedy) vinegar ; mucilaginous and oily drinks with opium. To prevent collapse, use camphor and ether. Emetics and purgatives are contra-indicated.

POISONING BY CORROSIVE ACIDS (*Sulphuric and Sulphurous*).

Symptoms.—Those of sulphuric acid poisoning are ulcerous stomatitis and pharyngitis, with violent gastro-enteritis, swollen and bleeding lips, eroded and much-inflamed mouth, slavering, difficulty of swallowing, violent colic pains, completely suppressed peristalsis, rapid and feeble pulse. Speedy death.

In poisoning by sulphurous acid (inhalation), one finds inflammation of the mucous membrane of the breathing apparatus, which varies much in intensity ; œdema of the lungs, with inflammation and emphysema.—*Autopsy* shows (in sulphuric acid poisoning), corrosion of the mucous membranes from the lips to the bowels, stomatitis, glossitis, pharyngitis, œsophagitis, gastritis and enteritis.—*Treatment :* diluted alkalies, soap-suds, lime water, dilute solutions of soda, potash and burnt magnesia. Opium along with mucilaginous and oily remedies ; stimulants (camphor, ether).

POISONING BY VINEGAR.

Symptoms.—Loss of appetite, colic, diarrhœa, staggering, stupor, twitchings, collapse, quickening of respirations, and pulse.—*Treatment :* alkalies, stimulants.

POISONING BY CARBOLIC ACID.

Symptoms.—Diminished appetite, slavering, vomiting, diarrhœa, slight colic pains, arching of the back; urine dirty, muddy, greenish-brown, containing albumen and smelling of carbolic acid. Paresis, and paralysis of hind legs, or paralysis of the entire body, sometimes sudden falling down, tremblings, timidity, restlessness, continuous convulsive spasms, stupor, coma, collapse. Fall of internal temperature with acceleration of pulse. Irregular and difficult breathing. Sometimes the symptoms of nephritis (white and red blood corpuscles, casts in the urine). Local corrosion when the acid has been concentrated. *Autopsy* shows: hæmorrhagic gastro-enteritis, fatty degeneration of the liver and kidneys, parenchymatous degeneration of the heart, and even parenchymatous nephritis. Dark, badly coagulated and greasy blood. Hyperæmia of brain and lungs, pia-œdema, serous discharge into the ventricles and a smell of carbolic acid in all the organs. Where locally applied, corrosion.—*Treatment:* sulphates, and particularly Glauber's salt, which neutralizes the acid. Soap, lime-water. Stimulants: ether, camphor, spirits of wine (alcohol), coffee, wine.

Poisoning by the Creosoles (creolin, lysol, bacillol, creosote).—The symptoms are almost identical with those of carbolic acid poisoning, viz.: trembling of the muscles, tonic clonus; sweating; weakness; staggering; sometimes sudden fall to the ground; paralysis; cardiac paralysis (shown by rapid, imperceptible pulse and subnormal temperature); dyspnœa; dark-brown urine; albuminuria. *Treatment:* stimulants and sulphates.

POISONING BY IODOFORM.

Symptoms.—1. The *acute form* of poisoning manifests itself first in slight gastric disturbances (loss of appetite, vomiting and constipation), then in sleepiness, stupor and coma, which are interrupted by fits of spasms. Dogs show sometimes at the beginning much excitement and even mania. Later, we find a great fall of internal temperature, and signs of heart weakness are developed (very frequent, feeble pulse, palpitation, dyspnœa, oliguria and albuminuria).

2. The *chronic form* of poisoning displays the symptoms of iodism, which are emaciation, atrophy of the glands, especially the lacteal gland, iodine-eczema and catarrh of the mucous membranes, particularly those of the nose (iodine catarrh), of the conjunctiva and of the mucous membranes of larynx and bronchi.—*Au'opsy* reveals: fatty degeneration of the larger glands of the body (liver, kidneys), as the most important change, also of the muscles of the heart and skeleton, with occasiona glomerulo-nephritis.—*Therapeutics*: It is, above all, imperative to remove the poison by the use of emetics, if this can be done; we have, however, no direct antidote. We may attempt to render it ineffective by administering strong doses ot starch flour. The treatment is, however, chiefly symptomatic and stimulative.

Chlorine Poisoning.—The symptoms here consist of severe inflammation of the mucous membrane of the upper air-passages, in coughing and extreme difficulty of breathing, with attacks of choking (Rost, Lungwitz). Acute gastro-enteritis with colic follows the internal administration of chlorinated lime (Beier).

Petroleum Poisoning.—*Symptoms*: Gastric disturbance, dizziness, stupor, appearance of intoxication, paralysis. The *treatment* consists in the administration of emetics and stimulants.

II.—VEGETABLE POISONS.

LUPINOSIS OF SHEEP.

Historico-statistic Notes.—Isolated cases of illness from eating lupine were already noted by Wienands, Liebscher, and Güttlich in the early sixties of the nineteenth century, but the first records of widespread sickness date from 1872. From this point, and especially since 1875, the affection of large numbers of animals became much commoner, so that the Prussian Ministry of Agriculture found it necessary to instruct the veterinary colleges of Hanover and Berlin to enquire into the nature of the sickness. Lupinosis is prevalent in the provinces of Pomerania, Posen, Brandenburg, West Prussia and Silesia, which, owing to the nature of their soil, are most suited to the cultivation of lupine. The disease has, however, spread into Hanover and is now found in nearly the whole of Northern Germany. In South Germany, however, the disease is a'most unknown. The injury inflicted upon agriculturists by lupinosis is considerable, for frequently from half to three-quarters of the affected animals perish. In the province of Pomerania the

losses amounted to many thousands a year. V. Below-Saleske reported that one district of the province lost in one year 14,138 out of a total of 240,000 sheep, besides being able to rear 13,000 fewer lambs than the usual average.

In addition to sheep, goats, horses, cattle, pigs and fallow-deer are subject to the disease. It can be transmitted experimentally to dogs, but not to rabbits.

Etiology.—Although similar symptoms had been noticed where no lupine had been eaten, yet true lupinosis occurs only where the plant is grown and used as fodder. It is especially the cultivated yellow lupine which is injurious, much less so that which bears blue or white flowers. Race, sex and age have no influence in predisposing to the disease; if lambs and ewes frequently suffer more severely than rams, the reason lies solely in their more delicate organisation. Various opinions have been given as to the exciting cause of the disease, but it is most probable that the injurious substance in lupine is a purely chemical poison. Evidence of this is found in the fact that the upper layers of stacks built in the open are found to be quite innocuous, the poisonous element having been washed out by the rain. A further proof is that the degree of illness is always in direct proportion to the amount of the food consumed, and that acute or chronic symptoms may be produced according to the quantity of the poisonous extract given; and lastly, that it is possible to extract the matter which produces lupinosis by washing the lupine in water containing 2–3 per cent. of caustic soda. Kühn suggested "Ictrogon" as the name of this extracted chemical matter. Arnold and Schneidemühl call it "Lupinotoxin," which is certainly a more appropriate name.

Older Theories.—1. Agriculturists at first ascribed lupinosis to a so-called "lupine exhaustion" of the soil. According to this theory, the plant, failing to find a sufficiency of normal nourishment in the soil, developed a substance possessing harmful properties. Lupinosis may, however, result from newly-planted lupines, and there are farms where lupines have been cut for fodder for many years from the same fields without the slightest ill-effect.

2. That the presence in the plants of fine dust or sand was a cause of lupinosis was disproved as early as 1876 by Dammann. In the course of his experiments he found poisonous lupines which were entirely free from dust and sand.

3. It has been suggested by certain veterinary authorities that the condition may be due to the presence of too much albumen in the blood, brought about by over-feeding with lupines. But it has been shown that poisoning may occur after the consumption of quite small quantities of the plant. Also, that on the same farm and in the same year, it may frequently happen that certain fields only, or even portions of fields, bear poisonous plants.

4. The affection of the plants by blight fungi was long accepted as the primary cause of lupinosis (Zürn). Many facts show that this theory is untenable; for it has never been proved that the pathological changes in the animal are caused by a fungus. It is well known that mouldy or even rotting lupines do not give rise to lupinosis, while it frequently happens that good plants in perfect condition, which have been carefully cut and dried, are highly poisonous. Where lupines are stacked in the open, the upper layer is often quite harmless, while the lower layers produce disease. This interesting observation in no way confirms the fungus theory. It is not possible to convey lupinosis by inoculation. The poisonous substance may be extracted from the plants by a 2–3 per cent. solution of soda, and the residue of this solution contains no trace of fungus. According to experiments made by Lemke and Arnold, lupines which had been exposed for four weeks to the action of absolute alcohol were still able to cause lupinosis. Had fungi been present, they would either have been rendered innocuous by the action of the alcohol; or they would have remained suspended in the alcohol, resuming their activity upon the introduction of the alcohol-residue into the animal. This, however, did not occur.

5. The theory that lupinosis is due to the presence of an abnormal quantity of the normal alkaloids is untenable. Brümmer and Krocker have proved that poisonous lupines frequently contain less alkaloid substances than non-poisonous lupines. And Liebscher has shown that the anatomical changes which arise in the course of lupinosis are quite different from those brought about by alkaloid poisoning, the latter being true nerve-poisons. Finally, Arnold has induced lupinosis by means of lupines from which the alkaloids had been extracted.

Properties and Preparation of Lupinotoxin.—It is only slightly soluble in water, but more easily in water made somewhat alkaline. Dry heat does not destroy the poison, even if raised to boiling point and maintained for three hours. On the other hand, steaming with 1–1½ atmospheres of high pressure for several hours very much weakens it,

and if the lupine be steamed with fully two atmospheres of high pressure the poison is entirely destroyed.

This poison lies chiefly in the pods, seeds and husks of the plant, but is also present in all parts. Arnold and Schneidemühl propose the following method of extraction :—Finely-powdered lupines are mixed with a 1½ per cent. solution of soda, heated to 105°–120° F., and stirred to a consistency of thick broth. After standing two days the liquid is pressed out. The filtrate is then evaporated in a water bath (140° F.), mixed with acetic acid, filtered and treated with a solution of acetate of lead, the filtrate saturated with sulphuretted hydrogen, evaporated at 120° F., and poured into 15 times its volume of 98° alcohol. The deposit is collected after 24 hours, dried and dissolved in water (it is a brown, resinous substance), mixed with acetate of lead and ammonia, and after precipitation, washed with distilled water containing ammonia, alcohol and ether, suspended in water and decomposed by sulphuretted hydrogen ; the precipitate is then evaporated at 160° F. and added to ten times its volume of 98 per cent. alcohol.

Respecting the generation of this poison in the lupine plant nothing certain is known. The possibility of its development as a result of metabolism by parasitic fungi on the lupines is, however, universally admitted.

Post-Mortem Appearances.—The principal changes detected on dissection of sheep suffering from this form of poisoning consist in parenchymatous inflammation of the internal organs, especially of liver and kidneys, with appearances of jaundice and a gradually developing hæmorrhagic diathesis.

1. The *liver* affection may be characterised as an acute parenchymatous hepatitis. A high degree of granular, albuminous opacity of the liver-cells is found, whereby the liver may be abnormally enlarged, or of its usual size, according as it has been previously affected by fatty infiltration. This initial granular opacity is quickly followed by fatty degeneration of the cells of the liver, which grow larger and contain larger or smaller drops of fat in addition to the granules of albumen. The intensity of this degeneration seems to depend upon the degree of nourishment of the individual. The liver becomes flabby, soft and friable. This stage of softening (at which most animals perish in a few days) is followed by atrophy (acute yellow atrophy of the liver), during which the softened fluid contents of the liver-cells are re-absorbed, and the liver itself grows much smaller. The stage of atrophy lasts about fourteen days. In chronic lupinosis these liver changes take the form of a chronic interstitial hepatitis, with new growth of interstitial connective tissue. Here the liver is smaller and firmer, sometimes lobulated and uneven, and the well-known phenomena of congestion are manifest (ascites, œdematous swelling of the spleen and intestinal mucous membrane).

2. *Jaundice*, which is generally present, affects first of all the liver, which is sometimes pale or citron-yellow, sometimes red or the colour of yellow ochre. The jaundice is hepatagenous, and produced by the enlargement of the liver and consequent biliary congestion between the cells, as well as by catarrh of the larger and finer bile-ducts. The liver may also show the discoloration of jaundice in isolated spots. In addition to the liver, the subcutis, the skin of the abdomen, the omentum and mesentery are also yellow. The gall bladder is often enormously distended and filled with bile, its mucous membrane being reddened and tumefied.

3. The *kidneys* display similar parenchymatous changes to the liver, but in lesser degree. Here, also, we find the signs of parenchymatous nephritis, consisting of granular opacity in the epithelium of the spiral tubes, accumulation of casts of exuded matter in Henle's loops, etc. In the urinary bladder, which is empty or nearly so, we find catarrhal cystitis.

4. In the *digestive organs* we notice, in addition to the jaundiced colour of the mucous membrane, a catarrhal and sometimes parenchymatous (glandular) gastritis, especially marked in the abomasum, with inflammatory redness and hæmorrhage in the small intestine, along with catarrhal affection of the entire intestinal canal.

5. The *heart muscle* is very pale and tender ; the blood within the heart and in the larger veins of the body is dark and thick, and coagulates quickly on exposure to air, turning light-red. In most of the organs (bowels, skin, subcutis, peritoneum, uterus and cerebral membrane) are found capillary hæmorrhages.

6. The *carcase* is generally much emaciated. The muscles greyish-yellow in colour, their fibres granular and in a state of fatty-degeneration, and sometimes having lost their transverse striation. The carcase decomposes quickly.

Symptoms.—Sheep, even when accustomed to being fed on lupines, dislike eating the poisonous variety, although they may do so at first, or after having been without food for some time. They soon, however, refuse entirely to eat the plant. Among the earliest symptoms is noticed a rise of temperature. This may even be observed one day after eating poisonous lupine, and in acute cases reaches 104-106° F., or even higher. The character of the fever seems, however, to be remittent, and before death the temperature sinks considerably.

The pulse is proportionately accelerated, reaching 130 a minute, or more.

Signs of jaundice may, in many cases, be noticed on the second or third day; in milder cases not until the fifth or sixth. They appear suddenly or gradually, and first, as a rule, in the eye. They may, however, be absent, and even the yellowish disco ouration of conjunctiva has occasionally been undetected by observers, from which one may conclude that jaundice is not necessarily a symptom of lupinosis.

As the disease advances, and indeed, sometimes from the very beginning, appearances of weakness and heaviness of the head show themselves in the animals; they manifest stiffness of gait, rise painfully to their feet, lie about and allow themselves to be freely handled. The heaviness of the head occasionally increases to insensibility: the animals hang down their heads, lean against the nearest support, or lie stretched at full length. They walk or stagger sideways, or backwards and forwards. They grind the teeth. The lower jaw is constantly agitated and the jaw muscles spasmodically contracted. Trismus frequently occurs, and they shrink with fear at the slightest noise.

Defæcation is at first usually retarded, the dung is hard and covered with yellow slime; later, it often becomes like tar and dark-brown, owing to admixture of blood. Now and then diarrhœa occurs. The urine, which is passed frequently in small quantities, is yellow in colour, containing mostly biliary pigment, and invariably bile-acids and albumen. It contains epithelial cells of the kidneys and bladder, round cells, as well as cylindrical urinary casts of various kinds.

Respiration is at first regular, but later becomes more rapid and painful (100 per minute). In some sheep a mucous discharge from the nose was oberved, as well as blood-stained foam.

A fatal end may follow in from one to two days, but the usual duration of the sickness is from four to five days. Death is caused by great emaciation and rapid failure of strength. Should the ailment tend towards improvement and recovery, the symptoms may pass away more or less quickly. At times the apparent improvement develops into cachexia.

The *prognosis* of lupinosis is very unfavourable. Only when the toxic effect is slight, and the offending fodder is promptly discontinued, is there any hope of recovery. Acute cases are generally fatal.

Treatment must be prophylactic, for up to the present no specific antidote to the disease itself is known. If, from economic reasons, it be impossible to entirely exclude lupines from the food, it is advisable to find out by experiment which fields produce the poisonous quality, taking each field separately, so as to avoid mistakes. This may be done as follows :—

1. Use the suspected lupine along with an excess of sound fodder, say, in the proportion of 1 to 6 or 10, and, if possible, for older animals only.

2. Allow the lupine-hay to be washed through in small heaps by rain in the open, and from larger heaps use only the upper layers for food.

3. Soak the injurious lupine in water, or better still, in a 1 per cent. solution of soda, for 48 hours, renewing the liquid from time to time.

4. Steam it for several hours under a pressure of two atmospheres.

5. Or place the lupine in thrice its bulk of water, to which 4 quarts of ammonia spirit have been added for each cwt. of lupine, and let the mixture stand two or three days at about 55° F. Afterwards soak in water for from seven to ten days.

If the sickness has already broken out, avoid the use of all alkaline mixtures, especially alkaline drinking water. It will be wise to add a little acid to the water, as it renders the lupine-poison insoluble. Furthermore, we recommend the use of evacuants, in order to remove the poison as rapidly as possible; but avoid Glauber's salt, which hastens solution, using rather oily remedies and the yeast of beer. The flesh of animals not too severely affected is quite fit for food.

Lupinosis in Horses.—As regards its anatomical changes and general symptoms this is similar to the same condition in sheep. It usually arises from the admixture of lupine, either in the fodder or litter. There is usually a more violent inflammation of the intestinal canal than is the case with sheep, and the first symptoms shown are sudden refusal of food, even of the best oats or meadow hay, abstinence from drink, dulness of the sensorium which may amount to extreme cerebral depression, during which the animal at times lays its head on the manger, grinds its teeth, hangs back with drooping head and displays an unsteady, staggering gait. The usual accompaniment of low fever may also become acute, the pulse rising to 60 a minute, or more. Breathing is generally steady, but may also be accelerated to 36–40 per minute. More or less intense icterus (jaundice) is present (conjunctiva being orange-red to citron-yellow, mucous membranes of mouth, nose and vagina clayey yellow). The tongue shows a coating of yellow mucus; evacuation becomes irregular, fæces in small balls, often covered with mucous and

very foul in smell. Urination, on the contrary, is frequent, but in small quantities. After these conditions have lasted for about eight days, an improvement sets in, and recovery follows in from 16–21 days. Fatal ending is unknown. Among unusual symptoms must also be mentioned ; slight attacks of colic, an orange-red and ropy nasal discharge, necrotic patches on the mucous membrane of the tongue about the size of a crown-piece, drying up of the skin over the bridge of the nose, skin inflammation accompanied by yellow, lustrous exudation, formation of crusts and detachment of the epidermis on the under lip and on the pasterns, with simultaneous œdematous swellings as far as the carpal and tarsal joints.

Chronic Lupinosis.—This arises from persistent feeding with only slightly poisonous lupine, and generally runs its course without jaundice under the aspect of chlorosis and cachexia. But nasal catarrh, conjunctivitis and, in solitary cases, inflammation of the skin and formation of scabs on the lips and ears have been noticed. Zurn also reports swellings on certain parts of the head (eyelids, lips, ears), with exudation of a yellowish fluid and subsequent formation of scabs.

Supplement to Lupinosis.—The characteristic signs of this complaint greatly resemble those of other, as yet unexplained, diseases, which have no connection with it. The most striking likeness is that between lupinosis and the so-called "typhous inflammation of the liver," or "malignant jaundice" of sheep after feeding with a large quantity of potato mash. The impression gained is that of complete identity between the two diseases. The complaint described by Sander as "liver-typhus in horses," and traced to feeding in inundated pastures, reminds us in all its symptoms of acute lupinosis. Reinemann and Jansen found that, under certain circumstances, eating of the straw of peas, beans and vetch may produce conditions like those of lupinosis. Stöhr noticed the same pathological changes in horses after feeding on vetch as in chronic lupinosis among sheep. In oxen, a dry necrosis of round patches on the skin was noted, and in horses and pigs, bowel inflammation. Wenke reports even fatal cases in horses after feeding on vetch (symptoms of paralysis). Mason found that after feeding on vetches four horses developed the following symptoms : inflammation of the hoofs ; amaurosis ; stiffness and tetanic rigidity of the limbs. One horse died 14 hours later with symptoms of lock-jaw.

The aspect of chronic lupinosis reminds us involuntarily of the so-called "Schweinsberger sickness," or "induration of the liver" in horses. (See "Liver Diseases," page 266.) Finally, there is so indisputable a resemblance between lupinosis, both acute and chronic, and the so-called clover-

disease of horses, that we consider it advisable to discuss the latter at this point.

THE SO-CALLED CLOVER DISEASE.

Etiology.—The injurious effects of an exclusive diet of clover for horses, and sometimes for cattle, arise, according to the unanimous opinion of authors, only from the use of bastard or Swedish clover (trifolium hybridum). The pathogenic properties of the latter have long been known. The true source of the trouble is probably to be sought in the mould (uromyces apiculatus ?), which appears to produce the symptoms detailed below, both by acute irritation of the gastro-intestinal mucous membranes, and also, indirectly, by its effect upon the brain and liver, after absorption of the toxic products of metabolism.

Symptoms.—These vary according as the skin and oral mucous membrane, or the internal organs be attacked. The changes consist of swelling of the fore part of the head, with excessive stomatitis, during which ulcers are formed within and without the cleft of the lips, as well as exudations from the epithelium of the tongue. Similar changes have been noticed on such parts of the head or limbs as are unpigmented. Here yellowish patches are formed, in some cases covered with blisters, which, on drying gradually, peel off and are very painful (similar appearances are noticed in lupinosis). Other general symptoms resembling those of lupinosis are yellow discoloration (like jaundice) of the oral mucous membrane and conjunctiva, attacks of colic, great faintness and somnolence, with nervous excitability (convulsions, raving, diarrhœa and attacks like those of epilepsy), staggering, paraplegia, amaurosis, paralysis of the pharynx. The course of the paraplegia, which is frequently enzoötic, closely resembles sub-acute inflammation of the brain, and is usually rapid and fatal.—*Treatment* is rarely attended by satisfactory results. The chief thing to do is to discontinue the offending clover. Other therapeutic remedies are merely symptomatic.

POISONING BY SHAVE-GRASS (*Equisetum palustre*) *limosum, arvense and hiemale.* THE SO-CALLED STAGGERING SICKNESS.

Etiology.—The toxicity of shave-grass, like that of lupine, varies according to climate, locality, etc. *Equisetum palustre*

appears to be particularly deadly. Of the nature of the toxin little as yet is known (alkaloids ?). Ludwig ascribes the toxic action of *equisetum* to the presence of acid aconite, but Lohmann and Reineck believe this substance to be neutral.

Symptoms.—At first, excitement and anxiety, the sensorium remaining unaffected; later, uncertainty of movement, reeling and staggering; finally, paralysis of the hind quarters, tumbling down, general paralysis, insensibility to external irritants, unconsciousness and coma. Pulse accelerated, appetite at first normal, but in course of time great disturbance of nutrition; sugar in the urine. Course sometimes very acute, death occurring in a few hours, but sometimes protracted (from two to eight days), and at times chronic (from one to several weeks). In cattle, after excessive eating, continuous diarrhœa becomes a prominent characteristic with the paralytic symptoms; while, if food be persisted with, cachexia and hydræmia, combined with weakness bordering on paralysis, make their appearance.—*Autopsy* reveals: hyperæmia, œdema, dropsical effusions on the brain and spinal cord, especially on the cerebellum; in cases of longer duration, hydræmia. Sometimes inflammatory changes in the mucous membrane of stomach and bowels.—*Therapeutics:* change of fodder, purgatives and stimulants, especially camphor; blisters along the spine.

POISONING BY BEECH-NUT OIL-CAKES.

Symptoms.—Very violent colic (intestinal tetanus), mania, fits of madness, continuous convulsive spasms which resemble those of strychnine tetanus, signs of paralysis, staggering, falling, with inability to rise. Course very acute, death from suffocation ensuing in a few hours.—*Autopsy* reveals conditions as in strychnine poisoning, sometimes red spots in the intestines.—*Therapeutics:* tannin, chloral hydrate, morphia.

POISONING BY CASTOR-OIL SEED-CAKES.

Symptoms.—At first gastric appearances: vomiting, difficulty of swallowing, colic, bloody diarrhœa and aneuria. Then weakness in the hind limbs, inability to stand, dulness of the sensorium.—*Autopsy* shows hæmorrhagic gastro-enteritis and sometimes also nephritis.—*Treatment* consists in adminis-

tration of mucilaginous remedies, opium, tannin and stimulants. Inoculation with castor-oil is recommended as a prophylactic measure (Cornevin).

POISONING BY COTTON-SEED MEAL.

Symptoms.—Only young animals are usually affected. They display disorder of digestive and urinary apparatus: tympanites, diarrhœa (afterwards bloody), hæmorrhage from the urinary organs, albuminuria, strangury and paralysis of the bladder. Duration is variable.—*Autopsy* reveals in the acute form hæmorrhagic gastro-enteritis, opaque swelling of the liver and kidneys, œdema of the lungs, dark-red urine, collection of liquid in the cavities of the body. There is no enlargement of the spleen. In chronic cases only, one finds general emaciation and dropsical phenomena, but at times, also parenchymatous nephritis.—*Therapeutics:* change of food, doses of castor-oil (for calves, 2–4 oz.; for lambs, 2–6 drams). Other treatment according to symptoms.

POISONING BY COLCHICUM AUTUMNALE (*Meadow Saffron*).

Symptoms.—At first those of acute gastro-enteritis: loss of appetite and vomiting, colic, violent and sometimes bloody and dysenteric diarrhœa, tympanites in cows, sometimes difficulty in swallowing. Increased urination, the fluid often being deep to dark-red (hæmaturia); later, when a nephritis has formed, anuria sets in. Dulness of the sensorium, apathy and somnolence to the extent of insensibility. Weakness, especially in the hind quarters; staggering, falling, inability to rise, tremblings. Small and, at times, imperceptible pulse; violent palpitation in horses; coldness of the extremities, sweating, livid discoloration of the visible mucous membranes, dilation of the pupils and accelerated respiration. Death occurs in from one to three days.—*Autopsy* reveals hæmorrhagic gastro-enteritis. Widespread ecchymosis, dark, thick and badly coagulated blood. Fatty degeneration of the liver (in one case on record).—*Therapeutics:* tannin, or remedies containing it, opium, mucilaginous drinks. In cattle, rumenotomy. Treat the gastro-enteritis and paralysis according to symptoms.

POISONING BY WILD POPPIES (*Papaver Rhœas*).

Symptoms.—Slavering, colic, constipation, tympanites, diarrhœa, intestinal bleeding; in cattle, raging fits of fury; bellowing, gnashing of teeth, desire to butt, biting, goring and wild dashing about. Also cramp resembling epilepsy, falling, unconsciousness, somnolence, tremblings and the appearances of drunkenness. These attacks last on an average several hours. The termination is, however, seldom fatal. Gerlach noticed also a chronic case of poisoning by wild poppies in horses after being fed with chop containing many of these flowers. The symptoms were those of chronic immobility, dulled sensation, etc.—*Autopsy* shows: gastro-enteritis, nephritis and signs of suffocation.—*Therapeutics*: tannin, cold shower-bath on the head; in the somnolent condition, stimulants: ether, camphor, etc.

POISONING BY TOBACCO (*Nicotiana*).

Symptoms.—Choking, vomiting, slavering, colic, tympanites, diarrhœa (intestinal tetanus); polyuria. Great muscular weakness, tottering, falling, inability to rise, paralysis and trembling of the whole body. Continuous convulsive spasms of the muscles, opisthotonus, cramp in the diaphragm, contraction of the eye-muscles, prolapse of the nictitating membrane, amaurosis, stupor and unconsciousness. The action of the heart is at first slower, but later becomes extraordinarily accelerated and very irregular, furious palpitation; coldness of extremities; difficult and even dyspnœic breathing. *Course*: If the poison is applied through the skin, is very acute, death happening even within an hour; it is generally acute and the animal may die within a day. Convalescence may last in some cases from eight to fourteen days.—*Autopsy* reveals hæmorrhagic gastro-enteritis (which, however, s absent when the poisoning has been through the skin); widespread ecchymosis of the subpleural and sub-peritoneal tissues, etc.; dark, fluid blood, hyperæmia of brain and lungs.—*Therapeutics*: Tannin, or remedies containing the same (which precipitate the nicotine); in cases of urgency, black coffee. Stimulants. In cattle, rumenotomy.

POISONING BY YEW-TREE LEAVES (*Taxus baccata*).

Symptoms.—Often sudden death resembling apoplexy, with staggering, falling and convulsions within a quarter of an hour. In other cases, the condition lasts longer (several hours to a few days), and is accompanied by gastro-enteritic symptoms : choking, foaming, vomiting, inflation, costiveness and frequent urination. Then stupor, tottering, falling, tremblings, spasms, the latter sometimes in the form of recurrent fits like epilepsy. Pulse small and slow ; cold extremities.—*Autopsy* shows gastro-enteritis (not constant), hyperæmia of the brain and œdema ; dark fluid blood.—*Therapeutics :* Often impossible, owing to rapidity of the course. In longer cases prescribe purgatives ; other treatment according to symptoms. In cattle, in case of urgency, rumenotomy.

POISONING BY CORN-COCKLE (*Agrostemma githago*).

Symptoms.—Phenomena of colic, vomiting, slavering, difficulty of swallowing, diarrhœa, regurgitation, paralysis, stupor, trembling, laboured breathing.—*Autopsy* shows intense gastro-enteritis, hyperæmia of brain and spinal cord, softening of the grey matter of the latter and appearances of suffocation. —*Therapeutics :* Change of fodder, purgatives, stimulants.

POISONING BY BOX-TREE LEAVES (*Buxus sempervirens*).

Symptoms.—Vomiting, colic, diarrhœa ; dizziness, stupor, appearance of being drunk, sometimes very speedy and convulsive death.—*Autopsy* reveals gastro-enteritis, with signs of asphyxia.—*Therapeutics* : Symptomatic ; tannin.

POISONING BY DIGITALIS (*Foxglove*).

Symptoms.—Slavering, retching, vomiting, colic, violent diarrhœa. Action of the heart is at first retarded, but later abnormally stimulated, palpitations, the beating of the heart gives a distinctly metallic sound. Pulse small, irregular and feeling as though it gave a double beat, finally quite imperceptible. Polyuria, albuminuria, strangury · excitement followed by stupor, faintness, staggering, paralysis of the under-lip, contraction of the pupils, spasms.—*Autopsy* reveals gastro-enteritis, endocarditis, myocarditis ; heart dilated, appearances of suffo-

cation (hyperæmia of the lungs and hypostasis, ecchymosis, dark, fluid blood).—*Treatment:* There is no known specific antidote, therefore this must be according to the symptoms, partly soothing, partly stimulative. Tannin may at least be tried. Chief stimulant, camphor.

POISONING BY OLEANDER (*Nerium oleander*).

Symptoms consist of vomiting, colic, diarrhœa, polyuria, palpitation; irregular, very feeble and retarded pulse; occasionally signs of excitement are noticed. Death follows with the appearances of paralysis (weakness, muscular tremblings). —On *autopsy* gastro-enteritis is found.—*Treatment* according to symptoms (emollients, stimulants).

POISONING BY LABURNUM (*Cytisus*).

Symptoms are slavering, retching, vomiting, sleepiness, staggering and general paralysis.—On *autopsy*, slight gastro-enteritis is found.—*Treatment:* Emetics, tannin; further treatment according to symptoms.

POISONING BY THE ERGOT OF RYE (*Ergotismus*).

Symptoms.—1. In acute poisoning, gastro-enteritic phenomena, which often show similarity to rinderpest; slavering, vomiting, colic, diarrhœa; ulcerous stomatitis like that of foot-and-mouth disease. Labour pains, abortion and even prolapse of the uterus with pregnant animals. Dizziness, dulness of the sensorium, insensibility to pain, paralysis, dilation of the pupils, cramp of the flexor muscles (ergotismus spasmodicus). 2. In chronic cases; necrosis of the extremities, such as the ears, tail, teats, claws and lower leg (ergotismus gangrænosus), sterility. Epzoötic abortion.—*Autopsy* reveals gastro-enteritis, signs of suffocation.—*Therapeutics:* Tannin, remedies such as will dilate the vessels, anti-spasmodic remedies; chloral hydrate and morphia.

POISONING BY PLANTS CONTAINING OIL OF TURPENTINE. (*The so-called Enzoötic Gastro-Enteritis.*)

Etiology.—Enzoötic gastro-enteritis is caused by the consumption of plants which are resinous or contain turpentine,

especially the young shoots of the coniferæ ; and also of astringent, irritating vegetable matter containing tannin (heather, bilberry plants, gorse or branches of the elder tree). We have, therefore, practically to deal with a case of poisoning by turpentine oil, with which all the symptoms agree. Enzoötic gastro-enteritis, first described by Chabert in 1787, is clinically to be regarded as a gastro-enteritis complicated by nephritis, which arises from pasturing cattle in the forests in spring time, or when fodder is scarce, and allowing them to eat the young fir-shoots and leaves. As this particular sickness was formerly very common, as compared with the rarer instances of poisoning, and has special aspects of its own, it was described as a separate disease under the above name. Now that forest pasturage is rarer, it is better to discuss it along with the toxic forms of gastro-enteritis, among which Delafond has already classified it. As the so-called enzoötic gastro-enteritis exhibits symptoms of hæmaturia, it has been usually described in literature under this name, and we shall therefore be obliged to refer to it again in the chapter on "Hæmaturia" and "Hæmoglobinuria." Here we propose, in the first place, to bring into prominence the gastric, or rather enteritic after-symptoms, as developed after eating matter which is resinous and contains oil of turpentine.

Symptoms.—This ailment, called in some places "forest or wood-sickness," is manifested by chronic gastro-enteritis, disturbance of appetite, fits of colic, constipation, restricted evacuation of dry dung covered with mucus or blood, fever, diarrhœa and gradually increasing emaciation and loss of strength. To these symptoms are added irritation of kidneys and bladder ; hæmaturia, ischuria, strangury and sensitiveness in the region of the kidneys. The condition generally lasts several weeks.

Autopsy reveals inflammatory changes in the stomach (especially the abomasum) and in the intestinal canal. These are the most important. There are also inflammation and degeneration of the kidneys, swelling of liver and spleen, and signs of suffocation. The carcase is generally very much emaciated.

Therapeutics: After removing the cause, we must treat these cases in general as simple gastro-enteritis. Administer mucilaginous remedies, stimulate the action of the skin by friction and hydropathic bandages. To counteract increasing

weakness and prostration, use stimulants. For the nephritic symptoms, tannin or sugar of lead may be given.

POISONING BY RAPE-SEED CAKE CONTAINING OIL OF MUSTARD.

Symptoms.—Chronic inflammation of the bowels. Tympanites, constipation, diarrhœa, bloody fæces, cessation of milk. Signs of brain irritation: thrusting, butting, turning; abortion. —*Autopsy* results: Hæmorrhagic enteritis.—*Treatment* according to symptoms.

POISONING BY CROTON OIL.

Symptoms are those of violent gastro-enteritis, with choleraic diarrhœa; general weakness and exhaustion; thin wiry pulse. Death occurs in from one to three days.—*Autopsy* proves the presence of severe gastro-enteritis.—The *treatment* consists in mucilaginous remedies and opium.

POISONING BY HELLEBORE.

(*Helleborus niger, viridis et fœtidus.*)

Symptoms.—Vomiting, colic, bloody diarrhœa; stupor, stupidity, weakness. Death occurs with spasms. *Autopsy* shows gastro-enteritis and the appearances of suffocation. *Therapeutics:* Symptomatic; use stimulants and especially camphor.

POISONING BY WHITE HELLEBORE (*Veratrum album*) AND VERATRIN.

Symptoms.—The same as in preceding paragraph, only that the gastro-enteritic phenomena are weaker, and where the veratrin is outwardly applied, are altogether absent. Chief symptoms are: violent vomiting and choking, slavering; severe pain; continuous convulsive spasms, which are even tetanic; great excitement, followed later by paralysis.—*Autopsy* shows all the signs of suffocation.—*Treatment* according to symptoms.

POISONING BY ACONITE.

Symptoms.—Stomatitis, slavering, retching, belching, vomiting, grinding the teeth, diarrhœa and very painful colic.

Inflation, weakness, paralysis, staggering, tremblings, convulsions, mydriasis, loss of consciousness and falling. Death in a few hours with spasms.—*Autopsy* shows gastro-enteritis and hyperæmia of the brain.—*Treatment:* According to symptoms; stimulants, especially camphor—also tannin.

POISONING BY RANUNCULUS (*Acris, arvensis, sceleratus*).

Symptoms.—Choking, vomiting, slavering, colic, diarrhœa, albuminuria; staggering, stupidity, tremblings and sudden tumbling. Death often resembles apoplexy.—*Autopsy* shows gastro-enteritis and signs of suffocation.—*Treatment:* according to symptoms, tannin.

POISONING BY THE PLANT MERCURY.

(*Mercurialis annua et perennis, or dog's mercury.*)

Symptoms are those of colic or gastro-enteritis; also hæmaturia, strangury, tenderness in the region of the kidneys; greatly accelerated breathing, rigors; small and rapid pulse. In one recorded case the symptoms of poisoning appeared ten hours after eating the herb.—*Autopsy* shows gastro-enteritis and nephritis.—*Treatment* must be guided by the symptoms (mucilaginous, anæsthetic and stimulating remedies).

POISONING BY EUPHORBIA (*Wolf's milk, or Spurge*).

Symptoms.—Colic, constipation, severe and even bloody diarrhœa, hæmaturia, tympanites, small pulse and palpitation, stupor (in cattle and sheep).—*Autopsy* shows presence of gastro-enteritis.—*Treatment* must be according to symptoms (opium and mucilaginous remedies).

POISONING BY HEMLOCK (*Conium maculatum*).

Symptoms.—Slavering, foaming, endeavours to vomit; stupor, staggering, paralysis, convulsions and rapid death.—*Autopsy* reveals nothing characteristic beyond the appearances of gastro-enteritis.—*Treatment:* Tannin, stimulants, especially camphor.

POISONING BY WATER HEMLOCK (*Cicuta virosa*).

Symptoms.—In addition to violent gastro-enteritic appearances, inflation, dizziness, stupidity, tumbling, actions suggestive of hydrocephalus, weakness, prostration, paralysis; muscular spasms resembling epilepsy, stimulated action of the heart and rapid convulsive death within 24 to 48 hours.—*Autopsy* shows gastro-enteritis, with signs of suffocation, œdema of the brain.—*Therapeutics :* Tannin, and narcotics according to symptoms, especially chloral hydrate.

POISONING BY FOOL'S PARSLEY (*Aethusa cynapium*).

Symptoms.—Slavering, retching, stupidity, stupor, paralysis, convulsions.—*Autopsy* shows gastro-enteritis.—*Treatment :* As for conium maculatum.

POISONING BY FLAX (*Linum usitatissimum*).

Symptoms.—Violent colic, inflation, diarrhœa, staggering, rigors, palpitation. Death with convulsions.—*Autopsy* reveals appearances of gastro-enteritis and the anatomical signs of asphyxia.—*Therapeutics :* Tannin, purgatives and symptomatic treatment.

POISONING BY NARCISSUS (*Poeticus and Pseudo-narcissus*).

Symptoms.—Intense gastro-enteritis and severe purging; stupor and debility; twitching and spasms.—*Autopsy* shows very severe gastro-enteritis.—*Treatment* according to symptoms.

POISONING BY PEPPER.

The *symptoms* are those of gastro-enteritis (in pigs).

POISONING BY ACORNS.

Symptoms.—Disturbed digestion; in severer cases gastro-enteritis, with persistent constipation, followed by diarrhœa resembling dysentery, tenesmus, passage of blood through the anus, passage of clear coloured urine in large quantities, great lassitude.—*Autopsy* shows intense gastro-enteritis, chiefly confined to the omasum; abrasion of the mucous membranes of stomach, etc.—*Treatment* according to symptoms.

POISONING BY ALOES.

Symptoms.—Colic, profuse watery diarrhœa, growing feebleness, faint, imperceptible pulse; death in from two to five days.—*Autopsy* shows gastro-enteritis; if long continued, the inflammatory signs may be absent, so that only the emptiness and paleness of the bowels are noticeable.—*Therapeutics*: Astringent, starchy remedies, and emollients.

POISONING BY MEZEREON, OR SPURGE LAUREL (*Daphne*).

Symptoms.—Stomatitis, slavering, colic, vomiting, diarrhœa, weakness; feeble, small pulse, etc.—*Autopsy* shows gastro-enteritis.—*Therapeutics* according to symptoms.

POISONING BY HORSE-RADISH (*Cochlearia armoracia*).

Symptoms.—Violent colic.—*Autopsy*: gastro-enteritis, with gelatinous infiltration of the walls of the stomach.—*Treatment* according to symptoms.

POISONING BY COMMON OR GREAT CELANDINE (SWALLOW-WORT). (*Asclepias vinetoxicum.*)

Symptoms.—Diabetes, strangury, general weakness and cachexia.—*Autopsy* shows nephritis and cystitis.

VARIOUS POISONOUS PLANTS.

Belladonna.—Mental excitement, raving, dilation of pupils, tympanites, paralysis.

Henbane or Hyoscyamus.—The same phenomena.

Thorn-apple, or Datura stramonium.—Appearances the same as in belladonna poisoning.

Sorrel (Common).—Gastro-enteritic appearances, especially diarrhœa; ater paralysis. *Autopsy* shows hæmorrhagic gastro-enteritis.

Rhododendron.—Slavering, choking, vomiting, colic, bloody diarrhœa, stupor, great excitability, paralysis.

Water dropwort (Oenanthe crocata).—Stomatitis, colic, diarrhœa, restlessness, bellowing, epileptiform and tetanic convulsions, general paralysis.

Cuckoo-pint, Wake Robin, or Lords and Ladies (Arum maculatum).—Dermatitis, shivering, dyspnœa, paralysis.

Lice-bane, or Louse-wort (seeds of the Delphinum staphisagria).—Stomatitis, colic, stupor, paralysis, anæsthesia.

Rough Chevil (Chærophyllum temulum).—Colic, stupor, general paralysis.

Common yellow Melilot (*Melilotus officinalis*).—Paralysis of muscles.

Male Shield Fern (*Filix mas; extract of filix*).—Vomiting, slavering, diarrhœa, loss of consciousness, uncontrollable movements, amaurosis, motor paralysis.

Common Bracken (*Pteris aquilina*).—Timidity, disturbance of consciousness, tottering, paralysis.

Cow-wheat (*Melampyrum*).—Sleepiness, cerebral depression, slight colic, muscular weakness, dizziness, bloody urine.

Poisonous Fungi—Toadstools, etc.—Maniacal phenomena, diarrhœa, salivation, staggering, and contraction of the pupils caused by *agaricus muscarius*.

Birthwort (*Aristolochia clematidis*).—Gastric phenomena, colic, polyuria, staggering, somnolence, imperfect vision, muscular weakness, loss of hair. *Autopsy* reveals stomatitis, gastritis and enteritis.

Polygonum.—Gastro-enteritis, paralysis, mental depression, nystagmus, etc.

Marsh Marigold (*Caltha palustris*).—Colic, tympanites, strangury, hæmaturia.

Broad-leaved Water Parsnip (*Sium latifolium*).—Gastro-enteritis, excitement, stupor.

Galega officinalis.—A case of poisoning in sheep is reported by Blanchard.

Potato-tops.—Symptoms of malt-eczema: mallenders, or scab-like eczema on the legs and genitals; stomatitis, diarrhœa, conjunctivitis. Appearances sometimes resemble those of foot-and-mouth disease. In other cases, symptoms resembling those of tympanites and intestinal catarrh have been observed, accompanied by reduction in weight and of the milk-secretion.

Ferula communis.—Bleeding at the nose, hæmaturia, hæmorrhage from the bladder and intestines.

Lily of the Valley (*Convallaria majalis*).—Symptoms similar to those of poisoning by digitalis.

Milk Vetch (*Astragalus mollissimus*).—Brain disorders, spasms, paralysis.

Garlic (*Allium*).—Apparent frenzy, slavering.

American Locust-tree (*Robinia pseudacacia*).—Colic, tympanites, weakness, paralysis.

Knotted Figwort (*Scrophularia nodosa*).—Diarrhœa, uncertain gait, lassitude.

Treacle-Mustard (*Erysimum crepidifolium*).—Choking, restlessness, staggering, convulsions, colic in geese.

Poplar (*Populus balsamifera*).—Colic, diarrhœa.

Common Viper's Bugloss (*Echium vulgare*).—Slavering, vomiting.

Hemp (*Cannabis sativa*).—Tyroler observed symptoms as of drunkenness in 25 steers after drinking water containing hemp (2 deaths). Mermelstein observed colic and staggering.

Atractylis gummifera.—Swine, sheep and poultry died after eating the thistles; the symptoms were those of tetanus and gastro-enteritis.

Pumpkin (*cucurbita pepo*).—After eating fresh pumpkin, some horses show decided dulness, others great excitement. The breathing is laboured the pulse-rate high; there is trembling, etc.

Andromeda florida.—Cases of sudden death among sheep are reported by Dougall.

Squill (*scilla maritima*).—Pigs died after eating squill (rat-poison), under symptoms resembling those of cerebral convulsions and erysipelas.

Prosopis juliflora.—So-called cashew-poisoning. Cashew-poisoning results from eating the fruit of the *anarcadium*, known as cashew-nuts. Prosopis juliflora is a stove-plant and a native of Jamaica.

Staves-acre (*Delphinium staphisagria*).—Wilcox observed poisoning by larkspur among sheep.

III.—ANIMAL POISONS.

POISONING BY CANTHARIDES.

Symptoms.—In the digestive organs: slavering, stomatitis, difficulty of swallowing, vomiting, colic, bloody fæces and diarrhœa with tenesmus. In the urino-genital organs: frequent urination with strangury, blood, albumen and casts; excited sexual impulse and redness of the vaginal mucous membrane. General debility, staggering, convulsions, falling; small, imperceptible, wiry and accelerated pulse. Sometimes conjunctivitis.—*Autopsy* reveals stomatitis, pharyngitis, even laryngitis and acute gastro-enteritis; nephritis; cystitis.—*Therapeutics*: Symptomatic treatment of the intestinal inflammation with mucilaginous drinks, opium. Stimulants, especially camphor. *No oil.*

POISONING BY SERPENT BITE.

(*Vipera berus and Redii.*)

Symptoms.—Lassitude, prostration, restlessness, shivering, dyspnœa, abnormally rapid pulse, which at last becomes imperceptible. The course is very acute. Local swelling, which creeps gradually towards the trunk.—*Autopsy* shows dark, fluid blood and local inflammation. No other sign is constant.—*Therapeutics*: Anti-venene, cauterisation by caustics, burning of the wound. Check absorption by application of an elastic bandage. Use spirits of ammonia both externally and internally, also subcutaneous injections of permanganate of potash, bichromate of potash, chlorine water, carbolic acid or creolin. Internally alcohol and other stimulants, especially strychnine and liquid ammonia.

Latterly, snake-venom serum and snake-gall have been recommended as means to obtain immunity and cure (Calmette, Fraser).

POISONINGS BY BEE-STINGS.

Symptoms.—These consist of local swellings of the skin, with a tendency to the formation of ulcers and necrosis, whereby sometimes considerable patches of skin become necrotic. More or less severe general phenomena are also seen, such as paralysis, stupor, septicæmia, hæmaturia. In cases of numerous stings in the region of the nose, suffocation may ensue as a result of the consequent swelling.—*Autopsy* reveals the symptoms of sepsis (fatty degeneration of the body muscles, clay-coloured liver, enlargement of the spleen, etc.).—The *treatment* is partly surgical (antiseptics, antiphlogistics, tracheotomy), and partly internal (stimulants).

Caterpillars and Grubs.—The swallowing of caterpillars and grubs in the food sometimes gives rise to stomatitis, gastro-enteritis, conjunctivitis, or dermatitis.

APPENDIX TO TOXIC GASTRO-ENTERITIS.

(*The Purely Narcotic Poisons.*)

Strychnine Poisoning.—Symptoms: are those of a rigid cramp (*Tetanus toxicus*); tetanic spasms, which last from a few seconds to several minutes, spreading over the whole body; stiff, outstretched attitude of the extremities, neck, vertebral column and tail; dyspnœa during the attacks; death by suffocation. Course very acute, averaging in dogs five to seven hours, after which time the danger is over. *Autopsy* shows no essential anatomical changes; signs of asphyxia. *Therapeutics:* The best specific is chloral hydrate (dogs two to ten grains), as well as repeated doses of chloroform carefully administered; also morphia injections and bromide of potash may be tried. Tannin, moreover, is also to be recommended as an antidote. At the outset emetics are advisable. Finally, artificial respiration may be resorted to.

Solanine Poisoning.—*Symptoms:* 1. The nervous form of solanine poisoning shows itself in staggering, stupor, paralysis, slower breathing; sometimes sudden fall and death in a few minutes; in other cases, the duration is from a few hours to one or two days. *Autopsy* displays no important results. 2. In the gastric form there is diarrhœa, vomiting, salivation, tympanites and sometimes stomatitis aphthosa. The latter is similar to that produced by saponin and occurs frequently after feeding on green potato haulms. 3. In the exanthematous form eczema appears upon the legs, nates, tail, udder and scrotum; conjunctivitis, blepharitis, and stomatitis are present. The exanthematic form is frequently complicated with the gastric variety.

Prussic Acid Poisoning.—*Symptoms:* Accelerated and laboured breathing, restlessness and fear; staggering, paralysis, stupor, shivering

twitching, spasms resembling epilepsy, vomiting, colic, diarrhœa and collapse.—*Autopsy* shows bright-red blood in acute cases, brown when chronic; smell as of bitter almonds; signs of asphyxia.—*Treatment*: Hydrated oxide of iron (see arsenical poisoning) acts as a direct antidote by formation of a non-poisonous salt; bleeding, artificial respiration, stimulants.

Poisoning by Charcoal Fumes.—*Symptoms*: Dizziness, stupor, loss of consciousness, asphyxia. *Autopsy* reveals bright-red blood and signs of suffocation.—*Therapeutics*: Fresh air, cold douches, blood-letting, stimulants.

Poisoning by Darnel-grass (*Lolium temulentum*).—*Symptoms*: In horses, chiefly cerebral; in cattle, loss of consciousness and general spasms. Sometimes appearances of colic are also noted. *Autopsy* shows slight gastro-enteritis; hyperæmia of brain and spinal cord.—*Treatment*: Tannin and stimulants.

Alcoholic Poisoning.—*Symptoms*: General excitement and restlessness; in cattle, even furious and maniacal signs. Increased redness of the mucous membranes of the head, palpitation, full and accelerated pulse. Later, stupor, staggering, symptoms of drunkenness, swaying about, total loss of consciousness, paralysis and collapse. Death not unfrequently results. Sometimes abortion and stimulated sexual desire. *Autopsy* shows no constant lesion. The organs smell of alcohol. Hyperæmia of the brain, and even hydrocephalus.—*Therapeutics*: Stimulants, especially coffee, ether, camphor, ammonia, carbonate of ammonia; cold douches on the head.

Poisoning by Chickpeas (*Cicer arietinum*) **and Chickling Vetch** (*Lathyrus sativus, Lathyrùs clymemum*). **Lathyrismus. Loco-disease.** —*Symptoms*: In horses roaring and difficult breathing, owing to paralysis of the laryngeal muscles; paralysis, weakness in the loins, suffocation. —*Post-mortem* reveals nothing of importance.—*Therapeutics*: Change of fodder; tracheotomy.

Poisoning by Common Gas.—*Symptoms*: Stupor, reeling, paralysis, convulsions.—*Autopsy* shows very light-red blood, hyperæmia of the brain. *Treatment*: Fresh air, artificial respiration, stimulants.

Chloroform Poisoning.—Suspended and retarded respiration; paralysis of the heart; in horses, occasionally, vomiting and inflammation of the lungs. *Treatment*: cease to administer the drug; fresh air, atropine, scopolamine.

Poisoning by Eserine.—Excitement, violent muscular tremblings, numbness, stupor, paralysis, weak action of the heart. *Antidote*: Atropine.

Poisoning by Pilocarpine and Arecoline.—Excessive secretion of perspiration and saliva; dyspnœa, mental excitement, paralysis.—*Antidote*: Atropine.

Poisoning by Morphia.—Excitement, spasms, constipation, coma. The administration of morphia and atropine in combination (paralysis of the shoulder) has been the cause of numberless fatal cases of colic in horses.

Poisoning by Apomorphine.—Excitement, spasms.

Poisoning by Santonin.—Restlessness, cerebral depression, staggering, reddish yellow urine.

Poisoning by Antifebrin.—Motor paralysis, cerebral depression, somnolence, palpitation, gastric disturbances, dark-coloured urine.

Poisoning by Naphthaline.—Hæmoglobinuria in horses; convulsions and mania in dogs and poultry; naphthaline cataract.

ANIMAL PARASITES IN THE INTESTINAL CANAL (*Helminthiasis*).

General Remarks.—The gastro-intestinal canal is in our domestic animals frequently the abode of bowel-worms (entozoa). Not all of these, however, are specially or demonstrably injurious to their hosts. The damage they cause is, first of all, dependent upon the numbers in which they congregate, and upon their species. On the other hand they often create abnormal conditions through injury to the intestinal walls and by their strange wanderings (*e.g.*, nematoids in the ductus choledochus). When present in great number they cause disturbance by irritation and by numerous, though small and insignificant, abrasions, as, *e.g.*, tænia echinococcus in dogs; they hinder the passage of the bowel contents, and may even, by knotting themselves together, produce complete stoppage (ascaris megalocephala in horses). Moreover, the absorption of material for their own growth and maintenance is harmful to the animal in which they live. On the whole, then, we may consider the following symptoms created by them as most important:

1. *Badly nourished condition (anæmia and cachexia).*
2. *Intestinal catarrh and hæmorrhage of the bowels.*
3. *Stoppage, as well as symptoms resembling colic.*
4. *Symptoms of brain irritation.*

There is nothing characteristic of intestinal worms in these symptoms, as they may all occur independently of them. They are, therefore, only to be attributed to worms when these are passed frequently, and, indeed, a positive diagnosis is possible only on the visible evidence of frequent passage of worms. We must not be content with such evidence, and neglect a careful examination of the sick animal, lest too hasty a diagnosis be made; for there may easily be other forms of disease present which are in no way connected with the worms, and

which may consequently be overlooked, to the injury both of the animal and its owner. Animals often manifest no signs during life which would lead us to suppose that they suffer from worms, and indeed appear quite healthy, even though worms be present in large numbers. The course of worm-sickness depends upon the kind and quantity of the worms in the bowels; serious results, especially with young animals, may occur, owing to the condition of cachexia produced. Regarding preventive measures, we are unfortunately only in a few instances as yet able to protect the animals from the invasion of worms (viz.: tænia echinococcus, t. cœnurus, t. cucumerina and trichina spiralis), for in the case of most of them we cannot even suspect the presence of the eggs or larvæ in food or water. Sometimes we find not only one individual, but several, and even many species of worms as causes of illness. In such cases we have to deal with a combination, so that it is impossible to single out any one individual or species. Especially in dogs and fowls we find the most diverse kinds of worms.

Frequency of Intestinal Worms in Dogs.—The following statistics give exact information on this point. Out of 70,000 sick dogs received in the Berlin Canine Hospital during nine years, 1,266, or nearly 2 per cent., were treated for helminthiasis. Deffke found during his investigations in the Berlin Dog Hospital from 200 cases of dissection, that 62 per cent. of all the dogs in Berlin suffer from intestinal worms. Of these 54 per cent. showed tapeworms, and 32 per cent. round worms. The most frequent entozoa in dogs are in that city the tænia cucumerina. The percentage of the various kinds will be best seen from the following table:

Tænia cucumerina..	40 per cent.
„ marginata	7 „
„ serrata	5 „
„ cœnurus	0.5 „
„ echinococcus	1.0 „
Bothriocephalus latus	0.5 „
Ascarides	18.5 „
Nématode du rein	1 „
Dochmius trigonocephalus ..	4.5 „
Spiroptera sanguinolenta	2 „
Pentastomum tænioides	6.5 „

Schoene found by his investigations in Leipzig as follows: 1. Tænia (and entozoa in general) manifest themselves in the different races of dogs in the proportions given below. Hunting dogs were in 52.94 per cent. of cases infested with worms (T. serrata being most numerous). Butchers' dogs to the extent of 66.66 per cent (T. marginata being most prevalent). Yard dogs showed worms in 40.44 per cent. of cases (contained most

T. cucumerina). Dogs used for purposes of draught, 72.22 per cent. (T. marginata most numerous). Sheep dogs were 57.14 per cent. (showed T. marginata, serrata and cucumerina), and were the only dogs that contained T. coenurus (7.14 per cent.). Of pet dogs, 70.37 per cent. were the bearers of entozoa (having 36 per cent. of T. marginata and cucumerina and 15.74 per cent. of T. serrata). 2. Ascaris marginata were most numerous in pet dogs (42.1 per cent.). 3. Strongylus trigonocephalus were only found in pet dogs (5.2 per cent.). 4. Echinococcus polymorphus only in draught dogs (5.5 per cent.); cysticercus cellulosæ and hemistomum alatum likewise only in draught dogs (5.5 per cent. of each). 5. Pentastomum tænioides appeared to be most plentiful in sheep dogs (as many as 21.42 per cent.).

Krabbe found T. marginata in 75 per cent. of all (100) dogs in Iceland, but in only 20 per cent. of all (500) the dogs in Copenhagen. Zschokke experimented upon 177, and found that 36 per cent., that is 61 dogs, had *tænia*. Of these, 38 had *t. cucumerina*; 9, *t. marginata*; 7, *t. echinococcus*; 3, *t. serrata*; and 3, *t. cœnurus*.

Worms are very plentiful in *Poultry*. In 18 dissected fowls Lucet found tænia proglottina in 18, heterakis papillosa in 15, trichosoma collare in 11, heterakis inflexa in 9, tænia infundibuliformis in 7 and tænia cesticillus in 6.

I.—TAPEWORMS.

Varieties.—The following kinds of tapeworm are found in the bowels of our domestic animals:—

A. In Dogs.—1. *Tænia cucumerina*, the most frequent of all. Its larvæ were proved to be in the dog-flea (*pulex serraticeps*); 2. *Tænia serrata*, which comes from the cysticercus pisiformis of hares and rabbits (cysticerci in the liver, lungs and peritoneum, vulgarly called the venereal disease of hares); 3. *Tænia marginata* from the cysticercus tenuicollis of the ruminants and of pigs; 4. *Tænia cœrnurus* from the cœnurus cerebralis of ruminants; 5. *Tænia serialis* from the cœnurus serialis of hares and rabbits; 6. *Tænia echinococcus* from Echinococcus polymorphus of the herbivora and pigs; 7. *Bothriocephalus* species, which are rare. The larvæ live in various fish (pike, perch, char, salmon-trout, grayling, sea-trout and eelpout, or turbot).

B. In Sheep.—1. *Tænia expansa*, which produces the tapeworm plague in lambs, and is said to grow to 65 yards in length; 2. *Tænia ovilla*, very rare in Germany, but common in France and Italy.

C. In Horses.—1. *Tænia perfoliata* are most common; 2. *Tænia plicata*; 3. *Tænia mamillana*, very scarce, only about ½ inch long.

D. In Cattle.—1. *Tænia expansa*; 2. *Tænia denticulata*; 3. *Tænia alba.*

E. In Goats.—*Tænia expansa.*

F. In Cats.—1. *Tænia crassicollis*; 2. *Tænia elliptica*: 3. *Dibothrium decipiens.*

G. In Rabbits.—*Tænia leporina s. pectinata.*

H. In Poultry.—1. *Tænia infundibuliformis*; 2. *Tænia lanceolata*; 3. *Tænia cuneata*; 4. *Tænia exilis*; 5. *Tænia cantania*; 6. *Tænia crassula*; 7. *Tænia fasciata*; 8. *Tænia setigera* (geese); 9. *Tænia trilineata*; 10. *Tænia coronula*; 11. *Tænia anatina* (ducks); 12. *Tænia gracilis*; 13. *Tænia sinuosa*; 14. *Tænia meg lops*; 15. *Tænia conica*; 16. *Tænia imbutiformis* (ducks); 17. *Tænia tenuirostris*; 18. *Tænia Davainea* (*proglottina, cesticillus, tetragona, echinobothrida, oligophora, carioca, mutabilis*); 19. *Bothriocephalus longicollis*, etc.

Natural History.—Tapeworms (cestodes) belong to the group of flat worms and form colonies (strobiles), whose individual members develop by budding from the scolex, which is the "nurse," or "head" of the entire colony. The riper members, which are capable of reproduction, are called proglottides. The head possesses a retractile trunk (rostellum) and attaches itself by suckers and hooks (armed tapeworms) to the intestinal mucous membrane. Each proglottis consists of an external muscular rind with an outer skin (cuticula), and an inner, which contain the generative organs and the vascular trunk. The sexual organs are hermaphrodite and consist of uterus, ovary and yolk-sack, of seminal vessels and vagina on one hand, and of testicles, spermatic duct and cirrus on the other. The sexual opening is usually lateral on the free margin. The eggs, which are found in the uterus in extraordinary quantities, have a very hard, membranous covering in several layers, and contain a globular, bright embryo, furnished with four or six hooks; they are very resistant, but may be destroyed by drying. If these eggs in any way reach the stomach of another animal, an embryo with four or six hooks is developed which perforates the wall of the stomach and wanders into various organs, where, after losing its hooks, it becomes enclosed in a bag (encysted) and changes to a "bladder-worm" (cysticercus). In this "bladder-worm" the embryo has already become a scolex and the progenitor of a later colony. Should it reach the stomach of a congenial animal, the scolex is set free by the digestion of the bladder, fastens

itself to the side of the stomach and develops into a colony of tapeworms.

The mode of development just described, in which the bladder-worm only represents the larval stage, or the sexless forerunner of the tapeworm in its various habitats, has been experimentally demonstrated by van Beneden, Küchenmeister, von Siebold, Leuckart, Haubner and others.

History of the Development of Tænia Cucumerina.—This is very interesting. Melnikow maintained formerly that the larva of this tænia was contained in the dog-louse (*trichodectes canis*). Contrary to this we have already shown that the origin of this, the commonest of all tapeworms, must be another, seeing that these lice are seldom found in dogs. Grassi has lately discovered that the dog-flea is really the host which transfers the tænia cucumerina. The larvæ of tapeworms are also found in the human flea (*pulex irritans*). The embryos exist free inside the body of the flea, often as many as 50 in one flea. The dog is infected by swallowing the flea. Young dogs whose mothers harbour fleas show tapeworms five to ten days after birth. As fleas often change their residence by jumping, dogs may also be infected which are themselves free from fleas. The true preventive, then, against this tapeworm is the destruction of the fleas.

Tænia Echinococcus.—The investigations of Müller and Mangold appear to confirm the earlier supposition of Ostertag, that there are two kinds of T. echinococcus, corresponding to the two kinds of echinococcus (*cysticus* and *multilocularis*). The common tænia belonging to the E. cysticus sort has stout hooks, the rarer or multilocularis shows slenderer hooks and a globular accumulation of eggs.

Tænia Serialis.—Railliet has shown that, in addition to T. cœnurus, another tapeworm occurs in dogs, viz. : T. serialis. Its relative, the cœnurus serialis, shows an arrangement of heads in a row, and grows as large as a hen's egg, inhabiting the connective tissue of the muscles, the subcutis and subserosa (peritoneum) of wild and tame rabbits, hares, squirrels and beavers.

Pathological Changes produced by Tapeworms upon the Intestinal Mucous Membrane.—The presence of these worms in the bowels, especially when they only occur singly, often produces no catarrhal change on the mucous membrane, or only a very slight one. But in other cases very intense catarrhal and inflammatory effects are caused. Schieferdecker found on examining dogs infested by T. cucumerina, singular tunnels in the mucous membrane of the small intestine, through which the linked chains of tænia travelled like railway trains. These tunnels were formed by the interlacing and growing together of the intestinal villi

In older sheep a previous gastric weakness would appear to produce this pre-disposition. The tænia expansa is remarkable for its rapid growth. According to Spinola, lambs of four weeks may contain tapeworms 12 yards long, a statement which has led to the unfounded supposition of an ante-natal infection. The phenomena of tapeworm plague have often nothing characteristic, and are not to be distinguished from those of cachexia, viz., chlorosis, pale skin and conjunctiva, and light-coloured wool, which can easily be plucked out, and is poor in fatty matter. Later on, serious disturbance of digestion follows; appetite and rumination both become irregular; the young animals are retarded in their growth, appear dull and feeble, may easily be caught, linger behind the flock and lose flesh rapidly. The constipation which usually results causes slight signs of restlessness; they keep lying down and jumping up again, arch their backs, and make vain attempts to evacuate, hold up their tails, run suddenly away and are more or less tympanitic. Should the cachexia become far advanced, diarrhœic discharges ensue, with great prostration. In this condition the lambs are incurable and finally perish of exhaustion.

3. In *Poultry*, with whom tapeworms are most numerous, the trouble shows itself partly in gastric disturbances, especially increasing diarrhœa, with slimy and even blood-stained fæces, and partly in steadily persistent emaciation and slowness of growth, although the appetite is well maintained. The birds are sad and languid, have lustreless and ruffled plumage, which usually harbours a quantity of parasites (mites, etc.), have at times fits resembling epilepsy, and often stand for long periods as though asleep, and then, seeming to awaken, run about and feed. Gradually they cease to eat, grow very feeble, fall down and die very quickly. The sickness is at times almost epidemic. Friedberger describes an extensive outbreak among pheasants. Lucet noticed an enzoötic appearance of tænia setigera among geese. The symptoms were emaciation, fœtid diarrhœa, paralysis and rapid death, the mortality being 20 to 30 per cent. (In one goose, 600 individuals of the tænia setigera were counted. A similar outbreak among young geese caused by T. lanceolata has been described by Ellinger (emaciation, spasms as of epilepsy, extreme diarrhœa and mostly sudden death). V. Rátz observed an enzoötic chronic enteritis which caused serious mortality among fowls, and which was due to *Davainea tetragona.* Rüther observed

extreme emaciation and anæmia in a hen, followed by death, as the result of *Davainea mutabilis.*

Tapeworms in the *other domestic animals* (horses, cattle, goats, cats, rabbits) do not so readily affect the general health. Where they do, the appearances are essentially as above described. Mégnin and Waldmann report on cases of perforative peritonitis in horses caused by T. perfoliata. Hurlimann found immense quantities of the same tænia in a horse suffering from paralysis. Hendricks describes a case of enzoötic intestinal inflammation produced in foals by T. plicata. Haubold and Truelsen mention two cases of T. denticulata in cattle; the symptoms were emaciation and chronic tympanites. In cats an epidemic of tapeworm plague occurred in 1874 in the Black Forest (T. crassicollis), during which the field mice also disappeared (cysticercus fasciolaris), probably from the same disease. The ailing cats grew rapidly thin and finally perished. In several cats which died of gastritis, Zschokke found T. crassicollis in the stomach, and attributed death to their presence there, as in healthy cats which he examined they were always in the small intestine. Malin and Deich report on similar cases.

Diagnosis.—This can only be determined when proof is shown that tapeworms have been passed, and in sheep usually only by *post-mortem* examination. The phenomena of chronic intestinal catarrh afford us no certain guidance. Tapeworms are usually evacuated through the anus with the fæces, and are then found on the surface of the dung-balls singly or numerous, and often movable sections, which, in dogs, creep about near the anus, stick to the hairs and finally dry up to thin yellowish or whitish flat objects. More rarely larger or smaller sections of tapeworm are vomited. In consequence of the irritation produced about the anus by the proglottides, dogs frequently rub along the ground on their hind quarters to relieve the itching. In other complaints they attempt to gain relief in a similar manner. The dilation of the pupils, which has by many been considered a sign of helminthiasis, has no diagnostic importance.

Differential Diagnosis of the various kinds of Tapeworm.—On this point and for their classification we must refer our readers to the text-books of general pathology and of parasitology, and will here merely point out the chief microscopic differences of the species most commonly found in dogs.

1. *Tænia cucumerina:* Frequently appear knotted together, the

mature proglottides resembling pumpkin seeds, *i.e.*, elongated and elliptical in shape, reddish, with two sexual openings on each free margin at most $\frac{1}{12}$th of an inch wide, 2 to 4 inches long (rarely 8 inches), and moderately thick. Under the microscope reddish egg-heaps are seen (so-called cocoons).

2. *Tænia serrata:* The proglottides united to each other like saws (hence the jagged edges); one sexual opening placed alternately left and right on the free margin; members very long (up to ½ an inch) and broad (to $\frac{3}{16}$ of an inch); barrel-shaped; total length from 19 in. to 39 in., and up to 3¾ to 4¼ yards).

3. *Tænia marginata:* Proglottides joined together in squares, often broader than long, with straight edges and alternating lateral sexual openings. Total length, 2¼ to 5½ yards. These are the broadest, longest, and also stoutest tapeworms of the dog.

4. *Tænia coenurus:* Proglottides quadrangular, the hindermost oblong, and very long in proportion to width. Colour, white. Total length, 19 in. to 39 in.

5. *Tænia echinococcus:* Has three links only (seldom four); about $\frac{1}{8}$ *of an inch long; only the last link or member contains eggs; links are* white, elliptical, and may be mistaken for intestinal villi.

These various proglottides may be microscopically recognised by the form of the uterus, and their heads by the differences in shape of their hooks.

Therapeutics.—There are many remedies in use for tapeworms, but all of them have to be administered in relatively large doses, and if they do not themselves possess purgative properties, no time must be lost between loosening the adherent heads and driving out the entire colony, as otherwise the scolices will re-attach themselves; an evacuant must therefore be given either with or soon after the anthelmintic. Treatment should be preceded by preparatory measures. These consist in fasting the animal for a day and emptying the bowels either by enemas or purging medicines, in order that the remedies may come into direct contact with the worms. All worm remedies should be given on an empty stomach. In the case of dogs it is well to have a second dose handy, as the first is often vomited.

1. *For Tapeworm in Dogs* use extract of male fern, 10 to 90 grains in capsules or pills, in the latter case generally with male fern powder, which may also be given alone (freshly powdered) in doses of from 2 to 6 drams. The extract of male fern is one of the most reliable tæniafuges. Then Kamala also has a good effect (½ to 4 drams), combining purgative with anthelmintic properties. Areca nut (2½ to 5 drams) grated in pills or mixed with fresh butter; kousso (2 to 6 drams) stirred up in milk and repeated in an hour; pomegranate root bark

(1 to 12 drams) boiled in water. Finally pumpkin seeds are also recommended (25 to 30 seeds), oxide of copper (1 grain), chloroform, benzine, creolin, lysol, and combinations of several tænicides, as areca nut with kamala, etc.

2. *In the Tapeworm plague among Lambs* it is first of all necessary to avoid all dangerous or suspicious pastures. All the remedies suggested for dogs may also be used here in appropriately modified doses. Of newer specifics, picrate of potash (7 to 20 grains) in pills, and also kussin (1½ grains) have been tried with good results. Others prefer for sheep the extract of male fern in doses of ½ to 1 dram, or kamala (1 to 1½ drams). According to Trasbôt the simplest and least injurious remedy is feeding with pine-tree sprouts (which contain oil of turpentine), twigs or berries of the juniper, all of which the young lambs eat greedily. At one time Spinola's worm-cakes were given. They contain tansy, wormwood and tar, 1 pint of each, salt ½ part, made into a cake with flour and water; also Chabert's oil (oleum contra tæniam, a distillation of animal oil and spirits of turpentine), which was given in tea-spoon doses with 3 to 5 grains of tartar emetic.

3. *For Tapeworms in Poultry* Gurn recommends powdered areca nut as the best remedy (30 to 45 grains made into pills with butter) but turkeys are said to be somewhat excited by it. Purgatives are seldom needed. Powdered tansy root (in doses of 15 to 45 grains) is said to be much inferior to areca nut. Mégnin recommends a mixture of ginger, gentian, fennel, aniseed, coriander and aloes (aä a pinch for each bird). Stiles prescribes copper sulphate with oil of turpentine, 1–3 teaspoonfuls. Railliet says that vermicides frequently fail with poultry.

All the above remedies may be used for the other domestic animals if required. For *horses*, the medicines mentioned in the next paragraph on the Ascarides are to be preferred, viz., tartar emetic, arsenic and spirits of turpentine, and areca nut in addition (two dessert-spoonfuls with each feed).

II.—ROUND WORMS OR ASCARIDES.

Natural History Notes.—Among ascarides in our domestic animals we must specially mention the following :—

(*a*) In *Horses and Asses :* Ascaris megalocephala.

(*b*) In *Dogs :* Ascaris marginata and ascaris mystax.

(*c*) In *Cats :* Ascaris mystax.

(*d*) In *Cattle and Pigs :* Ascaris lumbricoides.

(*e*) In *Poultry* : Heterakis (formerly ascaris) inflexa, and heterakis vesicularis, dispharagus nasutus in hens, heterakis maculosa in pigeons, heterakis dispar in geese.

The history of the development of ascarides is not yet clear. Formerly the opinion was held, based upon negative attempts at direct transmission, that they pass through the body of some intermediate host. Grassi has, however, lately proved by experiments in feeding, that the ascaris marginata is produced direct, without any intermediary, by ingesting the eggs from the fæces of animals afflicted with worms. This is confirmed by the observation also made by Grassi, that young dogs which were carefully isolated with their mother (already infected with round worms) were found to be infested with ascaris marginata, their only nourishment having been their mother's milk. We also have seen these worms in dogs a few weeks old, and Penberthy reports the same (a dog two weeks old was infested with ascarides; in another of six weeks 250 were counted). Liebenger found that all the calves of from three weeks to three months in certain stalls became infected with round worms. Railliet observed an epidemic of round worms among the 250 horses of a large livery stable, due to the eating of infected peat-moss litter. Epstein has observed the development of *ascaris lumbricoides* in the human subject as the direct result of swallowing ascarides ova taken from human fæces. Ten to twelve weeks later, fully developed ascarides were present in the subject, the eggs being found in the fæces.

Pathological Changes Produced in the Intestinal Mucous Membrane by Round Worms.—These worms, which are found sometimes in thousands, inflict severer injury upon the internal membranes than tapeworms, their heads being armed with toothed mouths. Indeed, they occasionally perforate the intestinal walls and produce peritonitis. Dogs may die suddenly from ascarides, and on *post-mortem,* we find on the inflamed and swollen membrane small, round, black-red points, which mark the places occupied by the worms, and show raised, wall-like edges with ulcerous indentations. In other cases signs of intense hæmorrhagic enteritis are found; occasionally the ascarides have formed passages and channels with ulcerous, eroded, and thickened walls in the mucous membrane. This process may extend through all the coats of the

intestine. The worms often knot themselves together in balls and completely block the lumen of the bowel. Several cases of perforation by ascaris megalocephala in horses have been described in which, at the point of attachment of the mesentery, small openings with thick, indurated margins were located, the wall having at this point less power of resistance. These openings led to bladder-like cavities filled with pus, dissolved food and ascarides between the two thickened layers of the mesentery. On perforation of these intra-mesenteric abscesses, an ichorous peritonitis was developed. Several observers have found a single round worm in the swollen and thickened orifice of the ductus pancreaticus. Ortman located seven in the distended biliary ducts of a pig. In poultry, the intestinal mucous membrane is more or less catarrhally inflamed, ulcerated and covered with thick, purulent mucus.

Symptoms.—Those caused by round worms differ only slightly from the signs of tapeworms. In general, they are disturbed digestion and nutrition, constipation, diarrhœa, tympanites, etc. ; emaciation and certain symptoms of nervous irritation—itching, rubbing, facial contortions, etc.

1. In *Horses* these worms often occur without visible symptoms. Or they produce only slight gastric trouble, such as diarrhœa and constipation, accompanied by intermittent colic and gradual wasting. Worms may, however, cause death by obstruction or perforation of the bowels. Occasionally they produce remarkable signs of distress. Thus, Freminet noticed a pronounced tetanus-like condition in a mare (trismus, see-saw movements, prolapse of the nictitating membrane), produced by reflex action, which vanished on the use of worm remedies. Truelsen saw epileptiform spasms in a mare, which also disappeared as soon as the worms were driven out. Damitz found, in one case, paralytic weakness of the hind legs, produced by a peculiar affection of the spinal cord, which rapidly ceased after administration of worm remedies.

2. In dogs, it frequently happens that the presence of round worms is not declared by any definite pathological symptom. Young dogs suffer most. The symptoms resemble those caused by tape-worms : chronic intestinal catarrh, emaciation, anæmia. Sudden death occasionally results both in dogs and cats from bowel-perforation. These cases frequently give rise to suspicion of poisoning until the *post-mortem* reveals their true state. In a few instances the worms pass through the gall-ducts, penetrate

the cæcum and colon), where, by means of their trephine-like armature of the head, they fix themselves. 2. *S. tetracanthus* becomes encysted in the submucosa of the bowels and wanders thence, fully matured, into the intestinal lumen.

Pathological Changes in the Bowels.—1. *S. armatus* causes, wherever it is fixed (by suction), bluish-red spots on the membrane, which may often be mistaken for ecchymoses, but under the microscope reveal a reddish worm, about $\frac{1}{50}$ to $\frac{1}{2}$ inch long, as their centre; a slight enteritis is also produced. 2. *S. tetracanthus*, when numerous, produces, particularly in foals, hæmorrhagic enteritis with minute submucous nodules (cysts), in the middle of which are yellow pus foci, with strongylus larvæ. Small scars are also often left on the mucous membrane. The worms are frequently found in masses in the intestines. Cobbold located sometimes more than 150 cysts per square inch of surface in the colon.

Symptoms.—Mature worms do not appear to affect the general health of horses, unless they are present in large numbers, when they excite a condition of the mucous membrane of the large intestine resembling dysentery, with consequent colic, which may even result in death. Violent straining and bloody, liquid excreta are noticed, and the arm is smeared on insertion into the rectum with a quantity of minute strongyli. Death sometimes follows from hæmorrhage of the bowels. Chronic cases occur occasionally, marked by much wasting and anæmia with severe diarrhæa. According to Schwarzmaier, diarrhœa is rife in wet summers among suckling foals, when death results from enormous immigrations of strongylus armatus. In England of late years, many fatal cases have arisen from strongyli, both in blood-horses and cart-horses (Walley, Penberthy). Heill reports cases in which meningitis and hydrocephalus internus with consecutive symptoms of immobility, were produced by worms penetrating to the surface of the cerebral cortex. Cansy speaks of atrophy and sclerosis of both testicles caused by invasion of those organs. Thrombosis of the left coronary artery, with sudden death, was observed by Cadiot in an ass; and hæmorrhage of the lungs in a horse by Michalik. Kitt and Blanchard found sexually mature strongyli in the peritoneum and pleura; Duncan the same in the abdominal muscles; and Pütz, Hinrichsen, Frohner, and others, in the abdominal cavity, within the tunica vaginalis,

and in the testicles of stallions and cryptorchids. Blanchard observed a case of fatal peritonitis resulting from perforation by a sub-peritoneal worm.

Treatment.—Owing to difficulty of diagnosis this is rarely possible. In general it is the same as for ascarides. Penberthy and Wilkie found oil of turpentine effective. Wilson recommends thymol. According to Spooner, palisade worms (strongyli) are difficult to drive out. Probably he alluded to the encysted larvæ of S. tetracanthus.

The history of the development of *sclerostoma armatum* is still somewhat obscure. Olt gives his investigations upon the wanderings of the strongylus armatus and the results of its depredations as follows: "The larvæ of the *Strongylus armatus* are probably introduced into the animal with the drinking water. They penetrate the mucous membranes of the cæcum and colon and work their way by preference into the sub-mucosa. Later, when they have attained sexual maturity, they return to the intestine where they copulate and attach themselves to the intestinal walls. The ova are released in the first stages of segmentation and are passed out with the fæces. In a few days, embryos emerge from the ova which follow the same course of development as that already described. There is no proof that the embryos pass into an intermediate, rhabditis-like stage (scolex). In penetrating the intestinal mucous membrane, there is danger of the larvæ finding their way into the blood-vessels, especially into the thinly-walled veins of the sub-mucosa, and of their being carried by the blood into various parts of the body. Once in the circulatory system they generally remain in the capillaries nearest to their point of entrance—those of the lungs and liver—where they speedily perish. Those which are carried into the larger arterial vessels settle in the intima, where they stay until they reach sexual maturity; while those which remain in the intestinal blood-vessels are able to return with the blood into the intestinal wall. There is, however, no certain proof that these larvæ perforate the arterial walls and work their way into the lumen of the bowel—in the horse. The number of strongylids present in the arteries is, as a rule, extremely small compared with those which occur in the bowels; it is, indeed, a factor of no importance in the maintenance of the species. Wherever *strongylus armatus* takes up its quarters, it gives rise to lesions in the tissues of its host which may lead to very varied results.

In its normal development the sclerostoma is a more or less harmless intestinal parasite. In the course of its wanderings into the sub-mucosa it does little harm to the bowel-wall, and remains in close proximity to its point of entrance. By the formation of a cyst at its seat of occupation, it protects its immediate neighbourhood from the depredations of other parasites, while the mucous membrane over the cyst gradually disappears, thus opening the way for the inevitable return of the worm into the lumen of the bowel. The individual sclerostoma which are carried by the blood into the capillaries, either die there or become so

effectually encysted by the reactive proliferation of tissue that they eventually cease to exist. "Entozoic nodules" in the liver and lungs of horses are generally brought about in this way, although they are frequently mistaken for glanders nodules.

2. Sticker distinguishes between three varieties of *sclerostoma armatum*, namely: *S. edentatum* (hookless); *S. bidentatum* (two-hooked); *S. quadridentatum* (four-hooked). The larvæ which occur in worm-aneurism are generally those of *S. bidentatum*. The passage of the young worms from the aneurism to the bowel is believed by Sticker to occur in the following manner: After the worms emerge from the larva-slough, they are carried by the blood into the end-capillaries of the mesenteric artery, where they lie in flat, knotted, mound-like thickenings of a size varying from a bean to an almond, until they attain the growth and development of sexual maturity. These entozoic embolisms are to be distinguished by their size and their situation in the media of the large intestine from the tumours formed by the *Sclerostomum tetracanthum*. The latter are situated in the mucous membrane; are much smaller (rarely exceeding a millet-seed in size); and they are not embolisms. The worms then enter the lumen of the bowel by crater-like openings in the mucosa; they attach themselves to the intestinal wall, attain full sexual maturity, copulate, and reproduce.

3. In the course of the *post-mortem* examination on horses conducted at the Pathological Institute of the Berlin Veterinary College from June to September, Willach found numerous worms in the large intestine, ranging from about $\frac{1}{4}$ to $\frac{1}{2}$ inch in length, which showed a striking resemblance to *sclerostoma armatum* in the formation of the head. Some, the females, had a band-shaped tail-like appendage; the rest a pouch (sac) exactly resembling that of the male adult worm, the external organs of generation being also in the main similar. But both types were sexually mature and in the ovarian tubes were numerous thin-skinned eggs. True males were not discovered. Willach assumes that these worms form part of the developing process of the *sclerostoma armatum*. He concludes, with Lenckart, that the sexually mature adult parasite gives birth to a swarm of rhabditides, which probably develop into the females and hermaphrodites described above, and which are both sexually mature. From the eggs of this intermediate form a new generation is hatched out which develops in a manner not yet known, into males and females, capable of reproducing their species. Willach denies that the passage of the parasites into the blood is necessary to their development. He considers it improbable that the rhabditides are able even to get into the blood-stream, as they have no mouths. Willach has also discovered an intermediate form of *sclerostoma tetracanthum*.

The history of the development of *strongylus tetracanthus*. Cobbold has thrown much light on the development of *S. tetracanthus*. The young worms, on quitting their cysts, form cocoons of mucous matter exuded from their skin, these may be found plentifully in the fæces of their hosts (as many as 40). The embryo forms in damp, warm media a few days after shedding this skin. The larvæ, which resemble rhabdites at this time, may live for many weeks in the open. But an intermediate host is needed for their further development. The fæces are ingested with green fodder or at pasture. They bore into the mucous membrane

of the cœcum and colon, to become encapsuled there after a second shedding of the skin. In time they break through their cysts and reach the lumen of the bowels as mature worms of both sexes. There they shed their skin for a third and last time and form the above-named cocoons. They attain their definite form and sexual maturity in the colon of their host. Von Rátz shows that the supposed finding of *anchylostomasia* by von Rathonyi in pit-horses was the result of confusion of the eggs with those of *strongylus armatus* and *tetracanthus*.

II.—STRONGYLOSIS IN SHEEP (*Strongylus contortus*).

Etiology.—The coiled palisade worms (*strongylus contortus*) which occur in large numbers in the abomasum of sheep, more especially of lambs and yearlings, and occasionally in goats, produces an affection which has been called the red plague of stomach-worms (red verminous gastritis). In economic importance it is not far behind the tapeworm plague. It occurs in spring and summer, spreads like an epidemic, and arises enzoötically in the neighbourhood of standing water, usually occurring with husk or hoose (strongylus filaria). From this fact, Gerlach concluded that they had a joint origin and sought to prove the statement. Strongylosis may occur after exclusively dry feeding, especially if the lambs are herded.

Post-Mortem Appearances.—Dissection shows the mucous membrane of the abomasum thickly covered with strongyli, whose intestines are red with the blood they have absorbed. The membrane is also in a state of chronic catarrh, and at times is perforated like a sieve. It must be noted that, owing to their being very quickly digested in the carcase, it is not easy to prove their presence some time after death. On the other hand, an autopsy is often necessary for the formation of a certain diagnosis.

Symptoms.—These cannot be differentiated from those of tapeworm plague. They consist in anæmia, chlorosis, pernicious anæmia with poikilocytosis, gastric attacks (especially diarrhœa), with an ever-increasing emaciation, weakness and cachexia.

Therapeutics.—*Treatment:* Turpentine, fœtid animal oil, or Chalbert's anthelmintic oil, picrate of potash (Rabe) à $1\frac{3}{4}$–$4\frac{1}{2}$ grs.; creolin and lysol (1 per cent. solution, 1 tablespoonful 3 times daily); areca nut (30–45 grs. mixed with bran to be given daily to each sheep for 4–5 days; after stopping the treatment

for a few days, it should be again repeated); extract of male fern (Moussu) and kamala. In the interests of prophylaxis infected animals should be isolated; infected pastures avoided, and marshy meadows drained; and good drinking-water should be provided (Stödter).

Strongylus convolutus.—Under this name Ostertag describes a new variety of strongylus which he discovered in the abomasum of cattle. Stiles mentions the same worm under the name of *strongylus Ostertagi-Stiles.* It occurs in the abomasum of 90 per cent. of all cattle. The mucous membrane of the abomasum becomes red and swollen in patches with grey spots, the size of a flax seed, which upon microscopic examination are found to contain coiled up strongyli. Ostertag says that when these worms are present in large numbers they give rise to cachexic dropsy, a condition which after slaughter is usually described as "dropsy of the tissues," or "dropsical condition of the flesh." According to Stadelmann, the worms make their appearance in October and November, and he found, in addition to *strongylus contortus* and *strongylus convolutus,* a new palisade-worm, the *strongylus circumcinctus,* in the abomasum of sheep. Stödler classified the worms found in the stomach of the domestic ruminants, and divides them into seven varieties as follows: *strongylus contortus Rudolphi, Ostertagi-Stiles, Curticei-Giles, oncophorus, Railliet, Harkeri, Stödler, retortæformis Ceder, filicollis Rudolphi.* He lays stress upon the fact that not only the *contortus,* but the other varieties also play a by no means unimportant part in the etiology of stomach-worm plague. McFadyean describes a verminous gastro-enteritis in cattle which was caused by *strongylus gracilis* as well as by *strongylus convolutus.*

In sheep, *strongylus hypostomus* sometimes gives rise to digestive disturbance with symptoms of colic. Upon examination, the small intestine will be found to be pricked all over with minute hæmorrhages, and the contents of the bowels to be chocolate-coloured. Bass observed an enzoötic occurrence in sheep, accompanied by diarrhœa and emaciation; there were fifty deaths.

Strongylus ventricosus was observed by Zschokke (Basel) as the cause of an enzoötic disease among the young cattle on a Swiss mountain pasture.

Strongylus rubidus is the name given by Hassall and Stiles to an unknown variety of worm which they found present in 25–75 per cent. of all the swine examined by them in Wash-

ington. The mucus from the bowels sometimes appeared to be blood-red from the number of worms which it contained. Marcone found *strongylus dentatus* in anæmic swine.

Strongylus cervicornis is the name given by McFadyean to a small nematode found in the abomasum of sheep and lambs, and giving rise to epizoötic diarrhœa. This worm is considered by other authors to be only one of the stages in the development of *strongylus contortus.* In the north of France and in England *strongylus cervicornis* has been recognised as the cause of epizoötic diarrhœa in calves.

Strongylus follicularis is a round worm, which, according to Olt, is the frequent cause of enzoötic chronic follicular ulcers in the colon and rectum of swine. These ulcers form the starting point of infectious intestinal inflammations, such as swine-fever, erysipelas, etc. Liebe believes Olt's parasite to be a young form of *strongylus dentatus.* According to von Linstow, it is identical with *œsophagostomum venulosum Rud.*

III.—DOCHMIASIS OF DOGS (*Dochmius trigonocephalus*).

Etiology.—This occurs especially among hunting-dogs which live in packs, and has been described by French and Italian veterinary surgeons under many names: dochmiasis, uncinariosis, ankylostomiasis, pernicious anæmia, bleeding of the nose, etc. This affection is common among all kinds of dogs in India. It is caused by dochmius trigonocephalus (*Uncinaria trigonocephalus*), along with which, dochmius stenocephalus is usually present. Besides these, trichocephalus depressiusculus is said also to co-operate. A similar parasitic anæmia has been observed in men (miners and workers in tunnels).

Autopsy.—The parasites are mostly found in the cæcum and small intestine. In slight cases, numerous dot-like hæmorrhages appear on the mucous membrane. In the middle or to one side of the small coagula several small, thread-like worms may be seen, ¼ to ¾ inch long, white, and with a black longitudinal line. In severer cases, the membrane is much thickened, and penetrated by fairly large blood foci. In prolonged cases, the dochmii are found singly, even in the colon, while their number in the small intestine has decreased. The carcase also shows signs of anæmia and cachexia.

(colic and prolapse of the rectum); into the bladder (colic, retention of urine); into the subcutis, etc. The adhesion of these larvæ to the pharynx and pharyngeal cavity cannot be designated a pathological condition, because they are often as harmless there as in the stomach.

In view of their harmlessness, treatment is generally needless, especially as the known worm-remedies have no effect upon them. Perroncito and Bosso recommend, as the result of experiment, carbon bisulphide, in doses of 5 drams, as the best remedy for horses. The beneficial effects of carbon bisulphide is confirmed by many (Buglari, Palenno, Cognesi, Hanke, and others). Wessel gives to foals of 1–2 years, 1¾ oz. in all; 2 capsules containing up to 93 grs. every 2 hours, and the next morning, 2–2½ drs. tartar emetic in the drinking-water. Larvæ are passed in large quantities for 3 days after this treatment, and the animal speedily recovers. When the larvæ invade the brain, treatment is generally impossible.

Larvæ of Œstrus in Dogs.—The larvæ of gastrophilus equi have occasionally been found in the intestinal canal of flesh-eaters, especially in the stomachs of dogs, where they hook themselves fast and flourish. French also noted an œstrus larva in the scrotum of a dog. Oestrus larvæ in the stomach of dogs are described by Mazzauti as gastrophilus canis (suspicion of rabies, symptoms of helminthiasis).

VII.—VARIOUS OTHER INTESTINAL PARASITES.

A. TREMATODES:—*Amphistomum conicum* in the first stomach of cattle, buffaloes, sheep and goats. Usually innocuous, except in Australia, where it appears to work great devastation among the herds and flocks. (*Amphistomum* occurs in cattle in Cochin-China).

Hemistomum alatum, in the small intestine of dogs.

Gastrodiscus polymastos, in the large intestine of horses, mules and asses, in Egypt and the Island of Guadaloupe.

Distomum echinatum (ducks, geese), *oxycephalum*, *ovatum* (hens, geese), *lineare*, *dilatatum*, *pellucidum*, *armatum*, *commutatum* (hens), *cuneatum* (peacock).

Monostomum mutabile (geese), *attenuatum* (geese), *verrucosum* (hens, geese, ducks).

The trematodes produce in poultry similar gastric disturbances to round and tapeworms. Treatment is also the same.

B. SPIROPTERA:—Among these we may notice:—*Spiroptera megastoma* and *microstoma* in the stomachs of horses. The

former cause nodules in the cardiac portion of the stomach, ranging in size from a bean to a walnut, each having an opening. These may set up gastritis and colic.

Spiroptera sanguinolenta, in the stomach and pharynx of dogs, in knotted clumps, may also produce gastritis. According to Grassi, the black beetle (*blatta orientalis*) is an important intermediate host of this worm. These beetles being very common in Southern Italy, the spiroptera also frequently infest the dogs there. Raillet states that the worms sometimes penetrate to the bronchial glands, to the lungs, into the walls of the aorta and even to the neighbourhood of the kidneys. In one case the bronchial lymph-glands were found greatly hypertrophied and infiltrated with purulent centres, containing spiroptera. The symptoms noticed during life were: difficult swallowing, vomiting, diarrhœa and extreme emaciation. In another case, a dog showed symptoms like those of rabies, and autopsy revealed worm-clusters in stomach, bowels and bronchial tubes, with inflammation of the duodenum. Künert, too, found this worm in knots the size of a walnut in the mucous membrane of the stomach of a dog which had been killed under suspicion of rabies.

Spiroptera strongylina, in the stomach of pigs. This worm, which was formerly considered to be harmless, locates itself under the mucous membrane of the stomach. According to von Rátz, it is sometimes the cause of severe epidemics among swine in Hungary. The disease, which is often fatal, runs a course of from 3–4 weeks; its symptoms are inflammation and ulceration of the stomach.

Spiroptera scutata, under the pharyngeal mucous membrane of cattle, sheep, goats and pigs (identical with *myzomimus scutatus Müller*, and *gongylonema pulchrum Molin.*)

Spiroptera pectinifera, in the crop of the fowl and guinea-fowl, is said by Neumann to give rise to a severe enzoötic disease, characterised by slow cachexia.

Spiroptera reticulata, in the peritoneum of the horse (von Rátz).

C. GNATHOSTOMA HISPIDUM the "hand-shield" stomach-worm of pigs. This worm produces verminous gastritis in pigs. Csokor found the serosa of the base of the stomach greatly infiltrated and covered with dark spots as large as poppy-seeds, the mucous membranes thickened to two inches and encrusted with small nodules. In it were forty worms, whose bristly heads reached as far as the serosa, where they caused the above-

CHAPTER IV.

DISEASES OF THE LIVER.

JAUNDICE. ICTERUS.

General Notes.—Jaundice like colic is not a disease of itself; both are merely symptoms of diseases. Authors have described:—1. A *catarrhal* jaundice, caused by catarrh of the biliary ducts; 2. A *hepatogenous* form, which sometimes appears as an accompanying symptom of liver complaints (parenchymatous, interstitial hepatitis, atrophy of the liver, congested and amyloid liver, fatty liver tumour-formations—such as carcinoma and sarcoma of the liver—distomatosis, echinococcus or hydatid sickness, thrombosis of the portal vein, etc.). To this latter form catarrhal icterus strictly belongs. 3. In contrast to these two forms of jaundice, which from their manner of development are also called congestive, resorption, retention, or mechanical icterus, and which are always referred to a disordered liver; there is yet a third form in which the yellow discoloration is traced to decomposition of blood in the process of septic, infectious or toxic diseases (sepsis, murrain, influenza, anthrax, poison, etc.). The existence of a true *hæmatogenous* jaundice has, however, been disputed. Of these three only catarrhal icterus can be treated as a separate disease, as in the two latter cases the icteric symptoms are secondary, and may even be absent altogether.

Etiology of Catarrhal Icterus.—This complaint is much more common among dogs, but occurs rarely with horses. Among 70,000 sick dogs treated in the Berlin Dog Hospital in nine years 102 suffered from icterus (0.15 per cent.). It arises from the swelling and consequent constriction of the mouth of the excretory duct, produced in the course of duodenal or gastro-duodenal catarrh, whereby the biliary duct and liver are surcharged with bile, which is reabsorbed by the liver and passed

into the blood. The causes, therefore, of catarrhal icterus are the same as those of gastro-duodenal catarrh, and consist mostly in errors of diet, the eating of damaged food; the influence of irritants (mechanical, thermal, chemical, or infectious) upon the mucous membrane of the small intestine; in congestion of blood caused by heart weakness; and in sympathic affection of the intestinal canal during feverish infectious diseases. In swine, jaundice has resulted in isolated cases from the penetration of round-worms into the biliary ducts (De Benedictis).

According to Bouchet, jaundice in the dog is not brought about by the inflammation and occlusion of the gall ducts as a result of gastro-intestinal catarrh. In two-thirds of the cases, catarrh was not among the *post-mortem* findings. He believes the condition to result from the presence in the gall-ducts and liver of the *bacillus coli*, accompanied by disintegration of the hepatic cells. He considers that the infection of the liver by the *bacillus coli* is brought on by chill, over-exertion, excitement, and bad food, and in jaundice recommends subcutaneous injections of caffeine.

Autopsy.—With the catarrhal changes in stomach and bowels, we find the mouth of the biliary duct opening in the intestine stopped up or constricted by the swelling of the duodenal mucous membrane. The membrane of the other part of the biliary duct is, on the other hand, seldom changed. The biliary passages are found much enlarged (we have seen one in a dog which was dilated to the thickness of a finger); the gall bladder is very full and distended, and even the smallest gall capillaries are distinctly injected. The bile, at the same time, often thickens to a crumbling, brittle mass. In the excretory bile-duct a firm mucous plug is sometimes fixed. The liver is enlarged, infiltrated with bile, and anæmic; the liver-cells are charged with pigment, and, if the disease lasts long, become atrophied (biliary cirrhosis). The bile can only be with difficulty removed by pressing the bladder.

All organs, except the peripheral nerves, the white matter of the brain, the spinal cord, and the tissue of the cornea, are more or less of a deep yellow colour. The carcase is anæmic. In the blood the white blood-corpuscles are more numerous, while the red vary greatly in size. Fatty degeneration of the myocardium is invariably present; the kidneys are anæmic; the epithelium of the kidneys in the extended tubuli uriniferi and loops has undergone fatty degeneration and is permeated

by yellow-brownish granular pigment. Finally, hæmorrhage is found in various organs, especially in their mucous membranes.

Symptoms.—The appearances of catarrhal icterus follow on those of gastro-duodenal catarrh. They are, therefore, generally preceded by disturbance of appetite, vomiting, coated tongue, increased thirst, diarrhœa, fever, etc. Then jaundice is developed, at first slight, but growing steadily more severe. The sclerotic conjunctiva and eyelids first show the characteristic yellow colour, varying shades from deep citron to orange. Later, the other visible membranes take on the colour, especially that of the mouth, and the skin (in sheep and dogs), also the urine, sweat, milk, etc. Specially characteristic is the dark yellow, or citron, or brownish-yellow, or greenish-yellow colour of the urine, which stains white blotting paper dipped into it, and may be extracted by shaking up with chloroform. It is due to the presence in the urine of biliary pigment in greater or less quantity. The chemical proof of its presence is best established by Gmelin's method with fuming nitric acid. This is slowly added in diluted form to the urine, when a change takes place wherever the two fluids are in contact from green to blue, violet and red. Of other methods, Fleischl's may be named, in which the urine is mixed with a concentrated solution of Chili nitrate of soda, and then with concentrated sulphuric acid. This also produces the same colour-changes. According to Rosin, a 10 per cent. alcoholic solution of tincture of iodine is sprinkled on the urine, on which a grass-green ring is formed round each drop as it falls. Besides colouring matter we often find albumen, though generally only in small quantities, also grains of pigment, drops of fat, diffusely-stained epithelia of the urinary duct, short urinary casts deeply infiltrated with pigment, and also biliary acids. In our chapter on gastro-duodenal catarrh we have fully discussed the fact that in dogs biliary pigments appear in the urine as a consequence of intestinal catarrh, yet without producing any discoloration of the mucous membranes; but in horses the reverse is the case, viz., jaundice of the membranes without the presence of biliary pigment in the urine.

While the absorption of these pigments into the blood leads to jaundice, that of the biliary acids in severer cases may result in much more serious symptoms. First it produces retardation of the pulse and fall of bodily temperature by several degrees

(to 97°, 93°, and even 90° F.), and by reaction upon the nervous centres, causes lassitude and weakness, dulness of the sensorium, and in horses frequently distinct symptoms of immobility. A further series of troubles is occasioned by the absence of bile in the bowels. For instance, constipation sets in, because the action of the bile on the peristalsis is lacking, and the quantity of fluid in the bowels is lessened by the same cause. The fæces are often highly offensive, their colour is lighter, and in dogs which are fed on meat are grey or clay-coloured, because the fatty matter is not extracted by the bile as usual from the contents of the bowels, and urobilin is consequently absent from the fæces. Enlargement of the liver rarely occurs in domestic animals.

The *duration* of catarrhal icterus varies according to the kind of animal affected. In dogs, owing to the total stoppage of the excretory bile-duct, the end is often fatal. In horses, on the contrary, the prognosis is more favourable. The disease generally runs several weeks. In the more severe cases with dogs (*icterus gravis*, *cholæmia*) hæmorrhagic diathesis sometimes ensues, with hæmorrhage in the skin, on the mucous membranes and in the retina.

According to Latschenberger, it is rarely possible to prove the presence of biliary pigment in fresh horse-urine by Gmelin's process, even when actually present; for it causes a dark-brown zone which hides the characteristic colour-change. He therefore first dilutes the urine freely with water, adds a solution of baryta and lets all stand from twelve to twenty-four hours; removes the muddy fluid above the precipitate and applies Gmelin's test to the latter. The colour reaction is, however, in this case only weak.

Therapeutics.—The treatment of catarrhal icterus corresponds with that of gastro-duodenal catarrh, and consists in strict adherence to a rational diet (flesh meat for dogs, green fodder, carrots, etc., for horses), and in the administration of anti-catarrhal salts, the best of which is artificial Carlsbad salt. Also aloes, castor oil, and tincture of rhubarb are recommended. Drastic remedies should be avoided, as they increase the swelling. Attempts have been made to reduce it by using tannin with red wine as an astringent, but with what success is doubtful. Mechanical manipulation is certainly more effectual. This includes (with dogs) emptying the gall bladder by pressure from the abdominal wall, and cold water injections into the rectum. Siedamgrotzky succeeded in faradising the liver transversely through the abdominal walls. Finally, Rancilla recom-

mends lead balls to remove the invagination, which is frequently present. To combat the weakness, somnolence and coma which occur during the latter days, and may end in death, stimulants must be used (camphor, coffee, ether).

The so-called "**Icterus of the New-born**" (*ikterus neonatorum*), often noticed in human beings, appears occasionally among domestic animals. Hartmann saw foals in Babolna in which jaundice was either present at birth, or after five or six days, and traces these cases generally to inflammation of the umbilical veins, with liver disease. In any case temporary constipation (non-delivery of the meconium) combined with weak blood-pressure may cause icterus in the newly born. The prognosis, according to Hartmann, is unfavourable, as most of those who suffer from it die in a few days from exhausting diarrhœa. He recommends alkalis with rhubarb as a remedy. Schöttler records a case where four foals of one mare developed jaundice upon the first day after birth. In the slaughter-house, calves are often found suffering from jaundice.

So-called **Icterus gravis** (*cholæmia*) in dogs is, in many cases, caused by complete stoppage of the excretory bile-duct by a mucous plug, or by invagination of the duodenum; less frequently, however, by pressure of the enlarged spleen, or by neoplasm of the pancreas. Or it may result from compression of the gall-ducts by carcinoma of the pylorus and duodenum (Eberlein). Here poison seems also often to co-operate, especially moulds, or phosphorus and common salt (bad meat, herring-brine). In other instances, acute bacterial atrophy of the liver seems, as in men, to be the origin. In a case observed by Schmidt, that of a circus horse, the left lobe of the liver became completely twisted round as the result of the horse rolling over. Cholæmia followed, ending in death. The course of cholæmia is extremely rapid, death following with sudden exhaustion and fall of temperature. The jaundice is very marked, the urine becomes dark citron-yellow, and the skin is also yellow, particularly on the lower parts of the body. Hæmorrhage may set in later on the mucous membranes.

RUPTURE OF THE LIVER (LIVER-APOPLEXY).

Etiology.—A rending of the liver with shattering of the hepatic tissue and hæmorrhage into the abdominal cavity may result from strong mechanical force. It occurs after a violent fall or wrench, *e.g.*, during colic, after contusion in the region of the liver, excessive work or over-exertion, such as leaping. Such mechanical force is not alone sufficient to cause rupture of the liver; for that organ must first be predisposed by certain changes of the parenchyma and vessels, or particularly by amyloid degeneration. (See paragraph thereupon.) Stress has lately been laid upon the connection between this last and rupture of o1 liver, on the ground that amyloid degeneration produces a

lessened power of resistance and greater friability of the vessels. In a similar way fatty degeneration of the liver induces rupture, as do also hyperæmic and inflammatory affections. Over-feeding may in lambs predispose to rupture by fattening the liver. Hæmorrhage of the liver occurs after embolism of the hepatic artery and obstruction of the hepatic veins in the form of so-called hæmorrhagic infarcts; during the course of infectious diseases and poisoning (anthrax and murrain, or poisoning by phosphorus) all of which result in fattiness of the liver-cells, or in parenchymatous hepatitis, and also in connection with certain new growths in the liver, *e.g.*, with cavernous tumours, melanomata and cancer.

Autopsy.—The *post-mortem* conditions in hæmorrhage of the liver are simple. When only superficial, the tissue of the liver is found to be softened and changed to a tender, blackish-red substance. The serosa is sometimes raised in the form of blisters above the peripheral blood-focus. In horses rupture occurs in the serosa, through which the blood gushes into the abdominal cavity, mostly on the convex side, and on the lower edge of the liver. The margins of the rupture are found covered with coagulated blood. When the hæmorrhage is more deeply seated the parenchyma is frequently infiltrated with numerous blood foci, which consist of thick blood and crushed liver tissue. In hæmorrhagic infarcts wedge-shaped, sharply-defined infiltrations are produced. The above-named degenerative changes may also be found in the liver. Liver-abscesses may also be sequelæ of hæmorrhage of the liver.

Symptoms.—In rupture of the liver these occur either suddenly, without premonitory sign, or in connection with certain pre-disposing liver affections, such as icterus, constipation, pain on pressing the region of the liver, etc. The aspect is precisely that of internal hæmorrhage :—The animals show great anæmia of the mucous membranes, restlessness, sweatings, cold extremities, weak and finally imperceptible pulse, with palpitation, staggering, falling, dilated pupils, rolling of the eyes, sudden blindness, amaurosis, etc. In severe cases of bleeding, death often occurs suddenly—in a few minutes. Slighter hæmorrhage may be resorbed, or lead to fatal peritonitis. In one case observed by Benjamin, of a horse kicked by another in the region of the liver, polyuria appeared in three days and lasted

for two months, *i.e.*, until death. On dissection the left lobe of the liver was found to be thickened, tender and infiltrated with an abscess.

Therapeutics.—When the course is rapid no treatment avails. In protracted cases absolute quiet, combined with the use of astringents: Ergot, sugar of lead, etc., may at least be tried. If weakness increase, as a result of loss of blood, we may also try stimulants (camphor, ether, alcohol, etc.).

PARENCHYMATOUS HEPATITIS.

Etiology.—Parenchymatous hepatitis, *i.e.*, inflammation of the secretory liver-cells, occurs in all domestic animals: Its causes are partly the same as those quoted for congestive hyperæmia of the liver, viz., damaged fodder, mechanical lesions, excessive heat, infectious disease, or poison. Clinically the disease is of subordinate importance, partly owing to its difficult diagnosis, and partly because it usually represents secondary changes in various infectious diseases or poisoning.

Autopsy.—The anatomical changes consist in enlarged liver, a bluntness of the edges, very soft and friable consistence, pale yellow, clayey colour, perihepatitis, adhesion of the lobes of the liver to each other, or of the liver to the diaphragm, enlargement and granular projection of the several acini above the surface of the rupture, opaque swelling, granular and fatty degeneration of the liver-cells, with pigment infiltration, hyperæmia of the interacinous connective tissue, as well as in formation of foci of softening, and of numerous small (or single large) hæmorrhagic foci in the parenchyma of the liver, or under the serosa, which may produce rupture of the liver.

Symptoms.—The symptoms of parenchymatous hepatitis are generally very vague. For this reason, diagnosis during life is difficult, and the condition can only be ascertained with certainty by *post-mortem* examination. Besides jaundice and signs of feverish indigestion in the horse, pain has been observed in the passage of fæces, caused probably by compression of the liver by the diaphragm and abdomen, as may be proved in smaller animals by pressure in the region of the liver.

Therapeutics.—Treatment consists in the use of antiphlogistic remedies, such as neutral salts, and in regulation of the diet.

Acute Yellow Atrophy of the Liver.—This is a very acute and pernicious form of parenchymatous hepatitis, and is most frequently observed in lupinosis, with which, in most of the cases described, it is probably identical. The so-called "liver-typhus," in sheep and horses, described by Haubner, Franzen and others, is generally an acute yellow atrophy of the liver, caused by mashed food, or by grazing in inundated pastures. Parenchymatous hepatitis also changes into acute atrophy after septicæmia, phosphorus poisoning, or after several infectious diseases. Autopsy reveals diminution (atrophy), softening and yellow discolouration of the parenchyma; the liver-cells show a fatty, granular degeneration. Extreme icterus of the liver (acute "yellow" atrophy), or, in many places, also considerable hyperæmia (acute "red" atrophy) may likewise be found. In the other organs various hæmorrhages appear. The symptoms are identical with those of lupinosis (gastric disturbances, icterus, cerebral symptoms). Thus, in horses, Adam noted at first, poor appetite, slight jaundice, reddish-brown urine; later, signs of depression, staggering, tremblings, slower pulse; and finally, intense icterus, with indications of immobility, accelerated pulse—all within from 25 to 48 hours. Similar cases are described by Stoll, Giancola and others.

Suppurative Hepatitis (*Liver-abscess*).—Abscess of the liver rarely occurs in the horse, but in cattle it is more common. The abscesses may have various origins:—1. Infection may come from the bowels, or metastasis from the arteries of the liver, *e.g.*, in pyæmia, glanders and other acute infectious diseases; 2. Thrombosis of branches of the portal vein or the umbilical vein (omphalophlebitis, so-called "navel-ill" in foals and calves); 3. Intrusion of foreign substances (fodder, barley-ears, sand, parasites), from the stomach, bowels or blood; 4. Mechanical injuries, inflammation proceeding from the bowels, gall-stones, etc. Hepatic abscesses are sometimes spread over the parenchyma of the liver in considerable numbers (during formation of metastasis), at other times singly, and are seldom confluent. Most of the pus foci are surrounded by a zone of thicker, dark-brown liver-tissue. Sometimes fully developed hæmorrhagic infarcts can be perceived, which are wedge-shaped, with central softness. In cattle we sometimes find ecchinococcus cysts which have discharged their contents. The symptoms of metastatic liver abscesses are uncertain, and the abscesses are often discovered only upon *post-mortem* examination. The chief characteristics are rigors, irregular and persistent fever, icterus and digestive disorder. At times they discharge their pus into the abdominal cavity, and more rarely into the portal vein. Albrecht describes a case of a horse where, in addition to fever, there was a cough.

Necrosis of the Liver.—Peculiar tumour-like nodules with necrotic centres, which have been termed necrosis nodosa multiplex hepatis, have in late years been found in cattle, pigs and sheep, and are traceable to intrusion of the bacillus of necrosis into the parenchyma of the liver. Nodular foci are found on the liver, which organ is much enlarged. These foci are limited in area, vary in size from a pea to a walnut, are round, hard and are found on section to be dry, spotted, grey-yellowish and often surrounded by capsules of hard connective

tissue. Their appearance is often accompanied by perihepatitis and peritonitis fibrinosa. Berndt, who observed 15 cases, all ending in death, says that the symptoms in cattle are clearly marked. The animals show diminished appetite, sluggish, stiff movement, undoubted enlargement of the liver, much sensitiveness in the region of that organ and of the entire abdomen, high fever (104–106° F.), constipation, strained breathing, inability to stand, and they finally die in from three to five days, after a prior appearance of liquid, tar-like diarrhœa, accompanied by sinking of the internal temperature. Berndt noted the complaint only in cows which were advanced in pregnancy or had recently calved. Patsche saw a case in a milch-cow which was not in calf. Werkmeister tells of a cow of which the liver had increased to three times its normal size and weighed 77 lbs.

Hyperæmia of the Liver.—The complaint is difficult to diagnose in our domestic animals, and its causes are manifold. We must not, however, confuse congestive hyperæmia with that arising from engorgement. The former is normal in every digestive process, and occurs in an abnormal manner in many disorders of the digestion, *e.g.*, after over-eating, or too little exercise (fat dogs), or after taking spoilt, irritant or mouldy food. It appears at the beginning of a liver-inflammation, after violent shock, excessive exertion, intense heat, or in warm, moist and swampy districts and steamy stables. Passive hyperæmia (caused by engorgement) is partly traceable to heart-failure (valvular defects, especially of the mitral); and to chronic lung-troubles (emphysema of the lungs, interstitial chronic changes of the lungs, compression of the same by hydrothorax, etc.). Hyperæmia of the liver occurs as a secondary symptom in infectious diseases, such as anthrax, inflammation of the adjoining intestinal canal, cachectic conditions, or intrusion of parasites into the liver. Autopsy shows the hyperæmic liver to be at first swollen and enlarged, darker in colour, roughly granulated (when broken) and relatively firm in consistence. The centre of its lobules is dark, their periphery light. If the disease lasts long, a so-called nutmeg liver results, in which, through the alternation of lighter and darker parts, it acquires the appearance of nutmeg. This arises from enlargement of the surcharged central hepatic veins, with consecutive compression, fatty degeneration, pigmentation and finally atrophy of the liver-cells. During the course of the chronic form, hæmorrhage may occur in the parenchyma of the liver with singular pigmentation of the same.

The *phenomena of hyperæmia of the liver* are so uncertain that it is rarely identified as a disease, but confounded generally with others and especially with gastro-intestinal catarrh. Of more important symptoms (which only too often are absent), we might name sensitiveness of the liver-region in dogs and a slight icterus, caused by compression of the bile-duct by the dilated hepatic veins when the liver is congested. But it is often impossible to decide whether it be a case of hyperæmia of the liver, or a hepatitis already developed. In intense hyperæmia, rupture and hæmorrhage of the liver may both occur. Treatment should consist in regulation of the diet, place of abode and exercise, as well as in the use of mild, opening salts, among which artificial Carlsbad salt deserves the first place.

Enzootic Hepatitis in Young Pigs.—Semmer describes an outbreak near Dorpat of hepatitis in pigs, the origin of which was traced to a certain micrococcus. Infection is said to occur after birth through the navel. The liver-changes produced are: enlargement, nodular surface, hyperæmia alternating with anæmia; and, on section, the appearances of "nutmeg liver," enlargement, fatty degeneration and pigmentation of the liver cells, with cellular infiltration round about them. The disease can be transmitted by inoculation to rabbits, guinea-pigs and white rats. Period of incubation, 20 to 74 days. Symptoms: Loss of appetite and lassitude, which only appear a short time before death. A similar condition has been observed by Chapius in sucking pigs the first few days after birth.

CHRONIC INTERSTITIAL HEPATITIS (*Cirrhosis of the Liver*). (*Chronic Induration of the Liver.*)

Etiology.—Chronic interstitial hepatitis appears as a slow inflammation of the connective tissue of the liver, with shrinkage and atrophy of the true secretory liver-cells. It occurs most frequently in horses as the so-called Schweinsberger sickness, also in dogs, cattle and pigs. Its causes are not yet known. The immediate one is probably to be sought in toxins generated by the intestines (food-poisons, auto-toxins, infective substances). Cirrhosis of the liver has been experimentally induced in swine by inoculation with the *bacillus suisepticus* (swine-fever bacillus). With horses, it is attributed to swampy country and irritant plants, whose poison is supposed to act upon the liver like alcohol upon that of man. In swine, alcohol itself may be an etiological factor, as it is by no means an infrequent food-constituent. Tschauner mentions the beer-dregs from restaurants. Among 350 pigs belonging to restaurant keepers in a certain town, thirteen had cirrhosis of the liver, whilst among 5,700 pigs from the country, three only showed any symptoms of it. In dogs, cirrhosis of the liver appears to be connected with valvu ar defects of the heart (engorged liver). In isolated cases among domestic animals, gall-stones may occur as a secondary symptom (Morel and Vaunay). [For cirrhosis of the liver arising from distoma, cp. Chapter on "Distoma Hepatica."]

Post-Mortem Appearances.—According to the stage reached by the disease, this reveals either a diminished and shrunken liver (atrophic cirrhosis), or one enlarged by new growths of connective tissue (hypertrophic cirrhosis). The con-

sistence is always harder and firmer, for, by the added connective tissue, the liver often becomes leathery or cartilaginous and creaks under the knife. In protracted cases, the surface is mostly granular, nodular and even lobulated; the serosa opaque and thickened. At first, the colour is that of a nutmeg, and later very light-yellow, while the entire organ is bloodless. On incision, a white, large-meshed net of newly-formed connective tissue is seen, more or less broadly streaked, which includes larger or smaller areas of fatty-degenerated and atrophied parenchyma of the liver, and separates the hepatic lobes by callosities of varying widths. Microscopically investigated, the process consists, at first, of infiltration of small cells in the periportal connective tissue and subsequent intra and extra-vascular development of similar tissue therein. Besides these changes in the liver we find hyperæmic engorgement and chronic catarrh of the stomach and bowels, enlarged spleen, ascites, anasarca, the cerebral membranes fully charged with blood, hæmorrhage in various organs, and in Schweinsberger sickness, excessive dilation of the stomach. More detailed information upon induration of the liver is given by Kitt and Rauscher.

Rauscher classifies induration of the liver in the domestic animals as follows: 1. Diffuse induration (porphyraceous liver; lead grey, enlarged liver in distomatasis; inflamed nutmeg-liver in Schweinsberger sickness); 2. Nodulous induration (knotted liver in cattle, lobulated liver in swine); 3. Granular atrophy (in dogs, cattle and swine); 4. Pitted liver (in cattle and sheep); 5. Nodulous fatty liver (dog).

Symptoms.—These develop slowly. The ailment generally begins imperceptibly with chronic digestive disorder, in which constipation alternates with diarrhœa, and the animal grows visibly thinner. Then follows slight jaundice, tenderness of the liver when pressed, and (in dogs and cats) ascites and anasarca. With horses, a peculiar dulness of the sensorium is superadded, with staggering and other signs of immobility, and also periodic fits of colic. The causes of these cerebral disturbances are obscure. They may lie either in re-absorption of the bile-producing elements of the blood (acholia), or in a complication with chronic pachymeningitis. At first there is no fever, but later on feverish symptoms arise—low to medium (103° F.). The duration is from a few months to several years. The disease is not unfrequently mistaken for immobility.

Therapeutics.—There is no cure known, but palliative treatment consists in the use of alkalies and neutral salts, especially Carlsbad salt, aloes and diuretics, with suitable diet, green fodder, etc.

Schweinsberger Sickness.—This ailment of horses, which reminds us in many points of lupinosis, is a peculiar form of chronic interstitial hepatitis. It was first noted at Schweinsberg, in the valley of the Ohm, in the Electorate of Hesse, and later, in many other parts of Germany. It is restricted to certain conditions of soil and of the plants growing thereon, being strictly local and enzoötic in swampy, peaty districts which are subject to frequent inundation. Probably it arises from the gradual action of a certain irritant poison, first upon the liver and later on the brain (after the analogy of alcohol, phosphorus or lupine-poison). The causes are, however, unknown. In the Glon valley, clover-feeding is spoken of as the origin; but farms lying on higher ground escape and horses of middle age (eight to ten years) are chiefly attacked. Later accounts state that hill-side farms and horses of all ages are subject to it, even when clover is not used for fodder, while the prevalence of the sickness is notorious; and in Schweinsberg it often carries off every horse in a stable within one year. The signs are at first unnoticeable, or mistaken for chronic gastro-intestinal catarrh. The animals have a diminished or debased appetite, lick and gnaw the woodwork, and neglect their fodder. They show dirty-red mucous membranes, yawn, are dull and depressed and have fits of colic. After weeks or months, jaundice sets in (but not always), the pulse is quickened, and peculiar attacks resembling immobility or staggers occur, to which are added periodical colic, constipation, complete abstinence from food, with medium fever. Emaciation keeps pace with the progress of the disease. Temporary signs of improvement are sometimes seen, especially after a change of pasturage; but the symptoms return with increased severity, and the strength fails rapidly, with œdematous swelling of the extremities. Death may follow in several weeks, or up to nine months, but rarely occurs in a few days. Occasionally rupture of the stomach causes a more sudden ending. The urine is strongly alkaline and contains albumen (Binhold).

Autopsy reveals the changes of the liver already described. These are principally an inflammatory hyperplasia of the interstitial connective tissue, especially that lying between the lobes, with localised fatty degeneration of the liver-cells. Sometimes the liver is double its normal size. Great distension of the stomach is also often found, as well as the aspect of chronic gastro-intestinal catarrh (slate-coloured discolouration and hypertrophy of the mucous membrane). *Treatment* is, as a rule, hopeless. Ganter recommends disinfection of the stable as a prophylactic. The intra-tracheal injections of Lugol's solution as recommended by Imminger and Huber have been found by other practitioners to be inefficacious (Putscher, Königer, Volz, Hillerbrand, Baer, etc). The animals, during the early stages of the disease, move about freely and show little sign of illness when at work, but are generally quickly sold by unprincipled owners, who know the hopelessness of the malady.

Smith describes a cirrhosis of the liver in American horses (South Dakota), which is known as "bottom disease" and bears a strong re-

semblance to Schweinsberger sickness. According to Gilruth, cirrhosis of the liver is common among horses and cattle in New Zealand. He states that he was able to produce the disease experimentally in two healthy calves of six months, by feeding them with rag-wort (*senecio jacobæus*). The animals died after four weeks. Guittard has frequently observed indurative hepatitis in geese fattened on maize. The symptoms are diarrhœa, loss of appetite, alcoholic composition of the excreta, and increasing debility.

AMYLOID LIVER.

Etiology.—Amyloid degeneration of the liver has so far been found in horses, dogs, cattle, sheep, rabbits and poultry, either alone, or in conjunction with amyloid kidneys, spleen or bowels. Its causes must be sought mostly in cachectic conditions, chronic inflammation, and permanent ulcerative processes. Rabe found in half the cases of amyloid liver in horses the simultaneous presence of chronic pleuritis, pericarditis, or peritonitis. He also observed it with hydrothorax and diffuse persistent formation of abscesses in the muscles. Caparini found amyloid degeneration of the liver in horses, conjointly with exudative pleuritis and increasing cachexia, combined with orchitis and phlebitis. Bruckmüller also identified the disease in cattle, united with chronic nephritis. Anomalies in feeding appear to be connected with its origin. Thus Bruckmüller noticed cases of amyloid liver among horses fed on malt and kept in very restricted quarters. The causes of amyloid degeneration of the liver are identical both in animals and man. Other recognised sources in man are : chronic phthisis, intestinal tuberculosis, chronic suppuration, ulcerous syphilis, bronchiectasis, dysenteric gastric ulcers, pyelitis, fistula, and ulcerous carcinoma.

Experimental Amyloid Liver.—By the repeated subcutaneous injection of cultures of *staphylococcus pyogenes aureus*, Maximow obtained in rabbits and fowls a rapid and extreme amyloid degeneration of various organs and of the liver in particular. This would indicate poisoning by pus-bacilli as the originating cause of amyloid degeneration. In a six-months calf, which had suffered from digestive disturbance for a few days, Hajnal found an amyloid liver as large as that of an ox.

Autopsy.—The liver is generally enlarged, even up to five times its normal size, and has thick and rounded edges. Its

consistence in horses, contrary to what is found in man, is soft, doughy, pulpy, or crumbling. Moreover, the otherwise smooth serosa shows in cases of longer duration an over-lying rind. The liver is greyish-brown in colour, and contains but little blood. The amyloid centres are sometimes evenly spread, and at others irregularly. In hens they also occur as disseminated nodules: Under the microscope the liver-capillaries, and especially the intra-acinous vessels, appear thickened, of homogeneous and glistening consistency, and crumbling away into flakes. This amyloid degeneration can also be demonstrated chemically. The parts just named take on a mahogany-red stain when treated with Lugol's solution of iodine, shading from grey to violet blue in dilute sulphuric acid and to aniline red in methyl violet. The true liver-cells share partially only in the amyloid degeneration ; but most of them, on the other hand, are atrophied and infiltrated with fat. In addition to the liver, amyloid degeneration shows itself in the spleen, kidneys and bowels.

Symptoms.—These are of a very uncertain character. A probable diagnosis can usually be made, if cachectic and anæmic conditions, combined with tumour of the liver and albuminuria (amyloid kidneys), be found. Icterus occurs only in isolated cases. The disease generally ends fatally in liver-rupture, with symptoms of internal hæmorrhage into the abdominal cavity.

Therapeutics.—Treatment is hopeless and can only be directed against the primary disease.

CANCER OF THE LIVER.

Pathological Notes.—This frequently occurs in old and fat dogs, rarely in horses or other domestic animals. It appears partly as a metastatic and partly as a primary cancer. The liver is then more or less enlarged, with an uneven surface and interspersed with nodules, ranging in size from a millet-seed to a man's fist. Sometimes these are isolated, at others confluent, or again are arranged in rows, and have usually a soft consistence, a pale yellow colour, and are fa rly rich in blood, with a soft or calcareous centre. Cancer nodules are then usually found in the portal system of the liver, as well as in the liver itself; also in the spleen, kidneys, pancreas, omentum, lungs, etc. Sarcomata and melanoma of the liver occur also and angioma (generally multip'e) are found in cattle.

Symptoms.—These are very obscure. Frequently they consist only in signs of gastro-intestinal catarrh or slight icterus. A probable diagnosis is possible only in dogs and cats by palpation of the liver-region, which sometimes reveals an irregular, lumpy surface of that organ, as, for instance, if an external growth of carcinoma be present (*e.g.*, on the mamma). Among consecutive symptoms of liver-cancer in dogs, is the development of ascites; and in horses the appearance of colic pains caused by peritonitis, and the addition of rupture of the cancerous nodules, which are frequently infiltrated with large blood-centres. This generally causes death by bleeding into the abdominal cavity. Extreme emaciation shows itself as the disease advances, even to muscular atrophy, which, in dogs, is most marked about the head. The symptoms in carcinoma of the liver are similar; Richter describes a case in a dog.

GALL-STONES.

Etiology.—The occurrence of gall-stones and their consequent morbid conditions (cholelithiasis) are, among animals, clinica rarities, although somewhat frequent among men. The explanation of this difference does not lie in any lesser predisposition on the part of animals to the formation of stones. On the contrary, experience shows that urinary calculi are found as frequently in domestic animals as in man, and especially in dogs. There appea then to be other reasons. From a dietetic standpoint we must remember that in man an irregular or abnorma manner of life and lack of due exercise occasion manifold disturbances in the region of the mesenteric vein, and may also be a predisposing cause of cholelithiasis. Pathogenic causes of this description are rarely active in the case of the domestic animals and are especially rare among the draught animals (horse). The fact that gall-stones occur so seldom in horses is partly due to the regularity with which they are fed and exercised; also to an anatomical peculiarity, namely, the absence of the gall-bladder in the genus equus. There is no doubt that the presence of the gall-bladder (man, dog, cat, ox) predisposes to the formation of calculi, because in this reservoir stagnation and decomposition of the biliary secretions may take place. Stagnation and decomposition of the biliary fluid being the two principal causes of cholelithiasis in man, it follows that in the human body, by far the greater number of gall-stones

are found in the gall-bladder. The stagnation of the biliary fluid, from whatever cause (catarrhal swelling, compression, occlusion of the gall-ducts, foreign bodies, parasites, low blood-pressure), gives rise, in the first place, to a thickening of the fluid and, later, to the deposit of salts and other substances contained in it (bilirubinate of lime, cholesterine). The decomposition of the biliary fluid (catarrh of the gall-bladder and gall-ducts) causes a change in the nature of its reaction, as in that of its chemical and physical properties. The result is that certain of its ingredients are deposited in an insoluble form, which, in the normal unchanged bile, would be held in solution (cholesterine, bilirubinate of lime).

Post-Mortem Appearances.—In the domestic animals gall-stones occur most frequently in the dog and the ox, and are found in the gall-bladder and the gall-ducts. Chronic interstitial hepatitis (cirrhosis of the liver) is not infrequent with them. The smaller calculi vary from the size of a poppy-seed to that of a pea. They are round, cylindrical, or egg-shaped, and are sometimes flattened at the sides. Faulkner found 87, Birnbaum 400 such stones in a horse. The larger stones may attain a length of six inches with a diameter of four inches. When found in the gall-bladder they are mostly pear-shaped, and cylindrical when located in the gall-ducts. The latter are frequently very much distended. As an instance, Messner found a stone in the gall-bladder of an ox which weighed 1 lb. 13 oz., and measured 3 in. by 6½ in. in diameter. Sometimes, in addition to one large stone, numerous smaller ones are found. Avérous discovered a stone as big as the fist, weighing 9 oz., in the biliary duct of a horse, in addition to thousands of other stones ranging from the smallest possible size to that of an almond. Trolldenier found a stone in a horse which was as big as the fist, brown in colour, rounded and nodulous, in addition to two dark green stones, weighing respectively ¾ lb. and 5 oz. Gall-stones have been met with in horses, swine and cats, as well as in cattle and dogs. Bursting of the gall-bladder, resulting from the accumulation of secretion consequent upon the occlusion of the gall-ducts by the impacted stones, has been observed in the ox (Cagny).

Symptoms.—These differ according to the size and position of the gall-stones. Such as remain in the gall-bladder, or very small stones elsewhere may cause slight digestive disturbance,

but no other visible form of illness. They are discovered rather as objects of interest during dissection. This refers to those cases of cholelithiasis in which only one hepatic duct is affected by stony formation. In man, even in cases where a hepatic abscess has formed, there are frequently no recognisable symptoms, or, at the most, they are very slight. True gall-stone colic, without jaundice, arises when a gall-stone becomes wedged in the bile duct. On the other hand, stones in the gall-duct, owing to the diameter of the latter, are less likely to give rise to symptoms of colic than to icturus gravis. Cases of gall-stone colic in the horse have been observed by Avérous, Trolldenier and Sommermeyer (mental lethargy, disturbed by periodic sudden attacks of colic, accompanied by stiffness of gait and dysuria). Marcus describes a case of gall-stone colic in cattle.

Therapeutics.—Treatment consists in the use of Carlsbad salts or of alkalies, in regulated diet and plentiful exercise. In the dog, as in man, laparotomy may be performed and the gall-bladder opened (Parascandolo).

PARASITIC DISEASES OF THE LIVER.—LIVER-FLUKE DISEASE.—DISTOMATOSIS.

Occurrence.—This disease is more common among sheep than cattle and goats. Pigs livers also often contain distoma. They are occasionally found in horses, buffaloes, deer, antelopes, camels, dogs, cats and rabbits. The disease is produced in sheep and cattle by two kinds of distoma, which occur, either singly or conjointly, in the liver bile-ducts and gall-bladder.

1. *Distomum hepaticum*, $\frac{3}{5}$ to $1\frac{1}{2}$ inches long, by $\frac{1}{5}$ to $\frac{2}{5}$ of an inch wide, leaf-shaped, occurs in sheep and cattle, also in goats and pigs, rarely in horses, asses, camels, rabbits and cats.

2. *Distomum lanceolatum*, much smaller, $\frac{1}{5}$ to $\frac{2}{5}$ of an inch long, $\frac{1}{25}$ to $\frac{3}{25}$ of an inch broad, lancet or tongue-shaped; appears in all of the above animals, except the horse and ass.

Natural History.—The liver-flukes represent the platyhelminths of the group trematoda. They live singly, and for their complete development have to pass through several generations and complicated metamorphoses. The following

are the several stages :—The ova are provided with a kind of lid, and pass out of the sheep with their fæces. In from four to six weeks, if there be sufficient moisture and warmth (development ceases in winter), they change into embryos furnished with a ciliated skin and a borer in front. These ciliated larvæ bore their way into certain snails, little shell-bearing limnæi (*limnæus truncatulus s. minutus*). The geographical distribution of the snail corresponds with that of the distomum hepaticum. They are very small, about $\frac{1}{5}$ in. long, with brown, spiral shells, exist all over the world, are very hardy and live more on land than in water. In this earliest host the embryo changes in fourteen days during summer (three to four weeks in winter) into a sporocyst (germinal bag), with germinative cells, which, by internal elongation, form so-called "redies" (tubes of immature distomata), and from these are directly produced (according to Leuckart)—but sexlessly, according to Thomas—the true distoma brood, or so-called tailed cercaria. They are microscopic creatures resembling tadpoles, which live freely in water, and of which about 1,000 spring from one egg. These still immature distoma soon become encysted by attaching themselves to grasses, etc.

Their ingestion by sheep follows consequently in the encysted state in the process of grazing. It may also occur through swallowing the snails or water containing cercaria. Thomas first proved that these tailed cercaria quit the bodies of their first hosts, and, after swimming about for a while, fix themselves to water plants or moist grass, where they shed their tails, roll up into a ball in a gum-like matter and thus form snow-white cysts, $\frac{1}{25}$ inch in diameter. In this state they can exist several weeks, and are eaten by sheep with the grass. That sheep sicken after eating snails containing cercaria has been definitely proved on experiment by Spinola, who ind ced the disease in a healthy sheep by feeding it with grass-snails taken from an infected sheep-pasture. In the Sandwich Islands the drinking-water is, according to Lutz, the principal, if not the only channel of infection. He says that the cercaria-cysts accumulate in the sediment of shallow brooks, that they are stirred up when the animals wade in to drink, and are then swallowed with the water.

The life history of the *distomum lanceolatum* is up to the present unknown.

General Remarks on Distomatosis.—The existence of this form of disease has been known for more than 500 years. In the old Germanic statutes it is referred to under the name of "Gewährsfehler" (breach of warranty), and was clinically described, as early as 1379, by the French sheep-breeder, Jean de Brie; by Hieronymus Gabucinus in 1547, and again later by Kornelius Gemma. The great enzoötics which broke out in Germany in the 17th century provided material for a more searching investigation of the nature of the disease. Pecquet (1688), Frommann (1676), Willius (1674), Wepper (1688), and others, wrote upon the subject at about this time. The writings of Leeuwenhoek, Schäffer (1764), Göze (1782), Chabert (1789), Bilhuber (1791), in the 18th century, deserve to be mentioned. The most important helminthological investigations of the 19th century are associated with the names of Mehlis, von Siebold, van Beneden, Eschricht, Steenstrup, Küchenmeister, Pagenstecher, Weinland, Zürn and Leuckart.

Distomatosis occurs enzoötically, and even panzoötically, in very wet seasons—so-called "snail-years"—which are especially favourable to the growth of the offending snail, but is rare in dry summers. It is frequent in damp districts, or those subject to floods. Hence it is endemic in districts which are liable to inundation, such as the Narenta valley in Dalmatia, as well as in the flat lands of Northern Germany, where the pasturage is marshy and fenny. In many places in Holstein, almost every slaughtered animal reveals traces of distomatosis. The years 1753, 1816, 1817 and 1854 are remembered as bad years; the seasons were very wet and the disease spread over large areas. Davaine reckons nine distoma years in the first half of this century for France; in 1812, 300,000 sheep perished in the neighbourhood of Arles (France) alone of this disease. In 1873 Alsace-Lorraine is said to have lost by this disease one-third of all its sheep, valued at £46,000. In Germany the fifties were years of the most serious agricultural losses, and in Ireland, in 1862, 60 per cent. of the sheep were attacked. In England the annual loss is estimated at one million sheep. In Slavonia, in 1876, 40 per cent. of all horned cattle are said to have perished from the ravages of liver-flukes. In South America in the district of Tondil (Buenos Aires), 100,000 sheep died in eight months of the disease. According to Popow, distomatosis was so prevalent in Poland in 1891, that on certain farms in some villages 50 to 90 per cent. of the sheep perished. He ascribes this loss to the increase in sheep-breeding as an industry

and the consequent use of low-lying pastures and of the hay cut from them. Krabbe says that *distomum hepaticum* is unknown in Iceland, though it occurs with great frequency in the Faroes. This is doubtless intimately connected with the condition of the mollusca of those districts. From the statistics of the Berlin abattoirs, it was shown that 36 per 1,000 of all cattle, 7 per 1,000 of all sheep, 2 per 1,000 of all calves, and 1⅛ per 1,000 of all pigs contained distoma.

The distoma brood is usually absorbed by cattle pastured in damp, swampy places, where the pools and puddles formed by continuous rain are particularly dangerous. The smaller spear-wort (*lysimachia nummularia*), a plant formerly singled out by shepherds, is not the cause of the disease. Lambs and yearlings sicken most readily, as well as animals of feeble constitution, especially fine-wool merinos. Infection may occur in the byre from eating green fodder, or drinking water which contains distoma. It may even possibly happen with dry fodder. It is interesting to observe how single animals sometimes escape disease. They may have been sick and not driven to pasture, or they may, perhaps, have fallen lame and been carried by the shepherd (Hahn).

Occasionally the disease seizes hares and deer, especially the females, which waste away to skeletons and perish. In the East Indies it is common among buffaloes.

IMMIGRATION AND EMIGRATION OF DISTOMA.

The liver-fluke does its work in the warmer months, unti the first severe frosts appear. It seems impervious to night frosts and may attack as late as December. Indeed, it is not improbable that, even in this climate, the ingestion of the liver-fluke may be active the entire year. Observation has proved that animals may quickly become infected, even after a quarter to half an hour's feeding in an infested pasture. Repeated invasions of the same animal may occur at different times, or of several animals in a herd at different periods. With regard to the mode by which the creature penetrates to the liver, the following three methods are possible :—

1. By the intestinal blood-vessels (branch-vessels of the portal vein), in the way that echinococcus larvæ are said by Heller to reach the liver. But proof of this supposition is lacking.

2. After losing through digestion their covering membrane, the distoma may perforate the stomach and small intestine,

and, by way of the peritoneal covering of the liver, reach the parenchyma, as well as the bilary ducts (Gerlach, Spinola, May). The occurrence of perihepatitis (peritonitis) has been adduced in proof of this; but it may also arise from centrifugal wanderings of the fluke within the liver, especially beneath the serosa or from perforation of the latter from within, outwards. Indeed distoma have been found with their heads projecting from the liver surface.

3. The distoma may move from the duodenum through the biliary ducts, and thus reach the liver. This supposition (Leuckart's) has most probability in its favour.

The further migrations of the distomum within the liver occur, according to Leuckart, with *distomum hepaticum*, as follows:—The cone on its head serves as an instrument wherewith to widen the narrower bile-ducts, while the scale-like points on its fore-part prevent any slipping back. Forward movement is procured by alternate application of its oral and abdominal suction-discs, with lengthening and shortening of the fore-part of the body. In *distomum lanceolatum*, which lacks these lateral bristles, progress is easier owing to its smaller size. The greater number of the flukes remain in the bile-ducts; others perforate the walls of the ducts by means of their head-cones and pierce into the parenchyma of the liver, which they destroy, perforating even the capsule as well, producing perihepatitis, or peritonitis. A third detachment reaches the branches of the portal vein, where it excites endophlebitis, thrombosis and emboli. Finally, a fourth party seems to attain the veins of the liver, and thence the more distant organs of the body. First, after passing the right side of the heart, they reach the lungs, where, as many have observed, they produce hæmorrhagic foci, passages charged with bloody matter, and even give rise to nodules in the lungs. In cattle, for instance, cysts or nodules have been found in the lungs, from the size of a hazel-nut to that of a hen's egg, which cysts show thickened and partially calcified walls, within which the flukes float in a brownish viscid fluid. Mackoldy found that either distoma or cysts containing 1–2 distoma (*pseudo-tuberculosis*) occurred in the lungs of 10 per cent. of all cattle slaughtered. Morot gives the proportion as 4 per cent. According to the latter, a bronchial distomatosis also takes place in cattle with accumulation of distoma and obstruction or dilatation of the bronchi. From the arteries of the lungs the flukes proceed into the veins, and through them into the general system of arterial circulation,

thus reaching every part of the body. Single distoma have been found in cattle in the sub-cutis, in the peritoneum, in the intercostal muscles, in the connective tissue of the retro-peritoneal space, and in the pancreas. In swine, they occur in the diaphragm and in the muscles of the larynx; in the horse they have been found in the scrotum on castration (Hiller). Lucet and von Rátz found distoma in the spleen of an ox and of a sheep. Von Rátz believes that, in these cases, the distoma migrated from the liver by way of either the sub-serous tissue or of the gastro-splenic vein. Peripheral abscesses containing distoma have also been met with in man; on the sole of the foot, on the occiput, behind the ear, and in the right hypochondriac region. The cases of sudden death from apoplexy, observed by Gerlach in the first stages of distomatosis in sheep, may be the result of embolism occasioned by the presence of young distoma in the blood-stream. According to Willach, the embryos of distoma have some connection with periodic inflammation of the eyes in horses and cattle. He also found cercariæ in the multiple embolic nephritis of calves, and assumes that, having passed from the bowel into the circulatory system, they were carried by the blood-stream into the kidneys.

The departure of the liver-flukes takes place when they are sexually mature, which occurs in about three weeks, according to Leuckart. Gerlach asserted that their exit takes place in from nine to twelve months after entrance, that is to say, from May to July. But Pech and Friedberger noticed great quantities of flukes in autumn and also in winter, located in the gall-bladder and duodenum. In the large intestine they are quickly digested, and are therefore never found. Thomas estimates the average life of distoma at over a year.

Post-Mortem Conditions.—The most important changes found in cases of distomatosis are, as might be expected, in the liver, and vary much according to the stage of the disease. The liver is enlarged, twice or thrice its normal weight, its margins blunt and the whole organ discoloured. The colour suggests porphyry, the ground being a dirty grey yellow or greyish brown upon which are irregularly scattered spots like islands, which vary from black-red to dark reddish brown in colour, and are either round or long in shape, being also sometimes clustered like branches. The upper surface of the liver is rough, uneven and covered with thread-like or membranous peritonitic pro-

liferations, which frequently cause its anterior surface to adhere closely to the hinder part of the diaphragm (*perihepatitis*). Sometimes holes are seen in the liver-capsule, from a pin-head in size to a millet-seed, through which a dirty bloody matter oozes, or the head of a liver-fluke protrudes. On cutting the capsule at this point, we shall often find beneath it a great number of young distoma, and, on their removal, sub-capsular cavities and hollow passages. The lymphatic glands on the hilus of the liver are œdematous, swollen and distended.

On section of the liver, in addition to serious changes of the biliary ducts, several void spaces of irregular shape and dimensions are noticed in the parenchyma, which are partially traversed by ridges containing a bloody, red-brown, or often greyish and pulpy matter, consisting of white and red blood-corpuscles, fatty degenerated liver-cells, waste material and a fluke, usually undeveloped. The biliary ducts are, as a rule, enlarged, often to three or four times their normal size. In cattle they may be as much as 1⅙ in. in diameter, and the liver appears as though inflated with air. In the ducts the bile is found red and slimy, and there are many distoma with their eggs. In the liver of a sheep as many as 1,000 flukes may be met with. The mucous membrane of the biliary ducts is at times in a condition of hæmorrhagic or mucous, or even ulcerous catarrh, owing to irritation by the distoma, and in later stages of the disease is thickened by new growths of connective tissue, often to an extraordinary degree. To this may be added an incrustion of calcium salts (phosphate of lime), which transforms the bile ducts into stiff, unyielding calcareous tubes, which can be felt from the outside and show prominently on the surface. Between these the true substance of the liver is more and more atrophied; it grows harder, creaks when cut and displays a lumpy surface (atrophy of the liver, cirrhosis). In the branches of the portal vein thrombi are sometimes found, produced by distoma moving freely in the vascular lumen. The gall-bladder usually contains a large quantity of muddy, dirty bile, single flukes and often many of their ova.

In addition to all these changes of the liver, ascites is constantly observed in severer cases, with isolated liver-flukes living free in the abdominal cavity, and transuded matter in the thoracic cavity and pericardium. The carcase is much emaciated, poor in fat, or else the fat is changed to a soft, gelatinous substance; the muscles are lax and pale; the blood

is thin and watery and the subcutis œdematously infiltrated (hydræmia and anæmia).

Symptoms.—1. *In sheep* there is nothing characteristic. Indeed, at first there are no signs of illness. The minimum duration of the latent stage is from five to six weeks, even with the most feeble animals. When, in the course of 1½–2 months, the changes in the liver have attained a certain degree of intensity, the symptoms which appear are those common to other diseases, such as severe cachexia, excessive disturbance of nutrition, as well as hydræmia and hydrops (so-called dry-rot of sheep). The sheep lose condition quickly, soon tire, show pale, washed-out mucous membranes, dry, lustreless wool which soon drops off, œdema on the eye-lids, on the larynx and hypogastrium. In cattle the coat becomes dull and ruffled, and the skin dry and adherent, and gastric disturbances supervene. The appetite is bad or depraved, rumination ceases, constipation alternates with diarrhœa, the urine becomes acid, etc. Sheep which are thus affected are very listless, linger behind the flock, can easily be caught, and their temperature shows many sudden and extreme rises. Later follow the symptoms of congestion : ascites, bronchial catarrh, etc. ; the conjunctival sac contains an accumulation of mucous secretion. Similar symptoms occur in goats.

2. *In cattle* the severer symptoms appear late, and the result is seldom fatal; It has been abundantly proved that in slaughtered beasts a very high degree of liver destruction may often exist without any signs having been noticed during life. (This is the opinion of an expert in forensic cases !) On the other hand, Weigel, Schwäbel and Thum assert that they have occasionally seen the disease end fatally in cattle.

Diagnosis.—Great diagnostic importance was formerly attributed to palpation for discovering enlargement of the liver, and to the jaundiced discolouration of the visible mucous membranes and of the skin. These signs are, however, usually lacking. Like Gerlach, we have never observed jaundice. Moreover, an enlargement of the liver beyond the posterior edge of the last rib, combined with greater sensibility of the organ, can probably be demonstrated only during the first stage ; for in more advanced conditions of the disease we have not observed it. More important for diagnosis is the micro-

scopical discovery of distoma-eggs in the fæces (oval and with a lid). In the greater number of cases no certain diagnosis is possible without actual *post-mortem*. We must not, however, forget that single distoma are extraordinarily common in perfectly healthy sheep and cattle.

Course.—This is chronic. Cases of apoplectiform death (embolus on the brain ?) are rare. According to Gerlach, the progress of the disease may be divided into four stages: 1st, that of traumatic inflammation of the liver—inflammatory swelling of the liver, generally not recognised during life and occurring mostly in the late summer; 2nd, the stage of chlorosis, from six to twelve weeks after pasturing on infected meadows, the chief months being from September to November; 3rd, that of emaciation, beginning a quarter of a year after reception of the fluke brood, the severest period of the complaint, marked by atrophy of the liver and formation of œdema; generally from January onwards. And 4th, the stage during which emigration of the distoma takes place, which Gerlach fixes at from May to June, but which may synchronise with the sexual maturity of the leeches, or about three weeks after their entrance.

Where the invasion is numerous, the course may be correspondingly rapid; but the minimum duration to death is never less than three months. On the other hand, the signs of chlorosis and wasting may, with older and stronger animals, pass away after a year, and recovery result. But this is often only apparent; permanent changes of the liver in various degrees always remain behind. In general the prognosis in cases of developed distomatosis is very unfavourable, and in this respect the winter and spring months are most dangerous.

Treatment.—As distoma in the liver are inaccessible, their treatment consists, above all, in prophylaxis. The chief points are, avoidance of the injurious meadows and destruction of the ova. Pastures become more dangerous as the damp weather sets in. As the shepherds' proverb says: "Suspected pastures must not be grazed after the feast of St. John." Care is most needful with lambs and yearlings. It is also important to seek for and destroy the snails in which the larvæ develop. The sheep should be kept well nourished, as they can then better support and resist the injuries inflicted. Thomas

recommends prompt slaughtering as the best means of destroying the ova, which is perhaps the wisest course. The livers should be buried, or destroyed by heat; but when boiled may be safely given as food to dogs. The affected animals and their droppings should be removed to dry pastures; wet fields should be drained and sprinkled with salt or lime; and rock salt should be supplied for the sheep to lick, in order to kill the immature distoma that have already reached the stomach. Such provision of salt may entirely supersede the bitter, aromatic or astringent remedies formerly employed. According to Frasbol, good results have followed from feeding the sheep with pine-tree shoots mixed with clover before driving them out to pasture. If the enzoötic spread of the disease cannot in any way be checked, the only course is to cease keeping sheep in the infected district. To prevent the introduction of the disease by strange sheep, Leuckart recommends the examination of their dung under the microscope, to see if it contain any ova of the fluke. Thomas says that sound meadows may possibly be infected by hares and deer.

The forensic aspect of distomatosis may be briefly summed up as follows: At German law, distomatosis in slaughtered sheep constitutes a *radical defect* when it gives rise to general dropsical condition. The period of warranty is 14 days.

Distomatosis in Other Animals.—A case in a she-ass is reported by Cadéac. The hepatic ducts were dilated like a bottle (ampulla). The animal grew very thin, showed great paleness of mucous membranes, and, on dissection, revealed ascites, hydrothorax and hydropericardium. According to van Vaelzen and Pease, distomatosis shows itself in buffaloes in loss of condition, stiffness of gait in the hind limbs, diarrhœa, tympanites, œdema of the skin and paralysis. It is much more destructive in the East Indies than in Europe. Common salt has been found an efficient prophylactic.

Other Distoma.—Besides the distomum hepaticum and lanceolatum the following occur: *Monostomum hepaticum* in the livers of swine; *distomum truncatum, albidum and felineum* in dogs and cats; (the latter probably become infected by eating fish); *distomum pulmonale* in swine, dogs and cats (America and Japan); *distomum magnum* in Texas cattle, in stags and antelopes; *distomum pancreaticum* in the cattle of Japan; and *distomum heterophyes* in the dogs of that country.

II.—ECHINOCOCCUS DISEASE.

General Remarks.—This disease is caused by the ingestion of echinococcus polymorphus (formerly echinococcus

veterinorum) (the bubble or cyst-worm), which is the larval stage of tænia echinococcus in the dog. It affects most frequently cattle, and after them, sheep, goats and pigs; rarely the horse, ass, cat, rabbit and dog; also men, camels and various wild ruminants (deer, chamois, antelopes, etc.), apes, various animals of the cat tribe, turkeys, etc. The echinococcus cysts have been found in almost every organ of the body, but their chief seats are the liver and lungs. They also occur in the heart, spleen, kidneys, the muscular system, the œsophagus, in omentum and mesentery, beneath the serous membranes, in the eye, brain, udder, and even in the bones themselves. Echinococcus sickness is widely distributed. According to Bollinger, it is in South Germany, with the exception of tuberculosis and distomatosis, the commonest of all infectious diseases among ruminants. Its geographical area corresponds with that of the dog. According to statistics compiled by Peiper from 52 slaughter-houses all over Germany, 11 per cent. of all cattle killed, 10 per cent. of sheep and 6½ per cent. of pigs suffered from this complaint. He states that the disease occurs with the greatest frequency among the cattle of West Pomerania and Mecklenburg (15–38 per cent.). In Greifswald, 65 per cent. of all cattle and 51 per cent. of all sheep are affected. The percentage of affected cattle in other districts is as follows: Hanover, Brandenburg, Silesia and Saxony, 7 per cent; Rhine Province, 2 per cent; South Germany, 0.1–3 per cent. Schmidt reckons the yearly loss on cattle in Prussia from echinococcus disease alone, at over £7,000. In the years 1888–90 the number of lungs and livers condemned in Berlin on account of echinococcus was: swine, 23,000; sheep, 17,000. Among 70,000 swine slaughtered in Moscow in 1890–94, 20,000 (that is 30 per cent.), contained echinococcus. According to Gurin, the highest percentage in Russia is to be found among the cattle of the steppes and is 82 per cent. In British India, where dogs are largely kept by the lower classes, echinococci are found in the livers of 70 per cent. of all cattle.

Natural History Notes.—The echinococcus cysts, which represent the larval stage of the three-segmented tapeworm of dogs, develop after consumption of tænia ova in the food, water, etc., and appear in four different forms—first, and most frequently, as a so-called *simple echinococcus*, consisting merely of a capsule, filled with fluid and containing brood-germs. Second—as *amalgamated echinococci*, in which the

principal cyst is broken up into so-called "daughter" (or secondary) cysts, or even into "grandchild" (or tertiary) cysts, which spring partly from the outer surface (exogenous), and partly from the inner surface (endogenous) of the parent cyst. Third—as so-called *echinococcus multilocularis or alveolaris*, a new growth resembling colloid cancer, which displays lacuna spaces on section, and which apparently proceeds from the expansion of an echinococcus which has no capsule, or has broken through its capsule, and develops mostly in the hepatic canals, and later closely resembles conglomerated tubercular nodules. On rare occasions it is as hard as stone. Fourthly—*echinococcus acephalus* (acephalo-cysts), which are headless and sterile cysts by no means uncommon.

The development of echinococci from the embryos, which probably migrate through the bilary ducts and portal vein system, is slow. In four weeks the. cyst is $\frac{1}{25}$ inch long, in eight weeks $\frac{1}{20}$–$\frac{1}{10}$ in. long ; after nineteen weeks it has attained the size of a nut, and only from five months do the brood-capsules, containing the head, begin to show. The true wall of the cyst is gelatinous, of lamellated structure, and consists of an outer and inner cuticle containing chitin. Around this gelatinous capsule is a solid capsule of connective tissue, which consists of granular matter, sparse muscular fibres and vessels. The echinococcus cyst, which ranges in size from a pea to a man's head, is filled with clear serous fluid, free from albumen. The largest ones hold as much as 10lbs. in weight. The scolices develop from the germ capsules (brood-germs) in the wall of the cyst, each of which may contain as many as 22 scolices, so that one bladder often produces 1,000.

Echinococcus multilocularis.—This is in general rare, but occurs, as Ostertag has shown, oftener in cattle than was supposed. During one year he found 30 cases in cattle in the Berlin abattoirs, only one in a pig (among 200,000), and none in sheep. In the slaughter-houses at Troyes (France), Railliet and Morot observed it in cattle and sheep in about 100 cases, generally in the liver. Grundelach found it in the breast-bone of a cow ; Wallay and Schmidt in the liver and lungs of sheep. The disease of men is distinguished from that of cattle by the fact that the latter causes no clinical symptoms. Ostertag considers it probable that the multilocular E. does not represent a mere variety of the ordinary E., but a particular species with an as yet unknown tapeworm. This idea has been confirmed by recent investigations of Müller and Mangold. Indeed, two sorts of tænia echinococcus are actually present in dogs. The common tænia E. unilocularis has clumsy hooks, and the tænia E. multilocularis much thinner ones.

Post-Mortem Appearances.—Livers affected by echinococcus cysts are usually more or less enlarged, sometimes even five or tenfold, and present a lumpy surface, corresponding to the cysts. Their weight is also proportionately increased. Thus a liver, whose normal weight is about 10 lbs. may be 150, or even 158 lbs. in weight. One such, weighing 132 lbs., contained 2,400 cysts. A pig's liver of 4 lbs. may grow to 50 lbs.; Perroncito found one of 110 lbs. The serosa of the liver is often thickened, and the liver itself firmly adherent to the neighbouring diaphragm, omentum or intestines (perihepatitis). The echinococcus cysts dislodge the parenchyma so that it is found on section, to be permeated by a number of cavities, between which compressed remnants are left, which are atrophied, insulated or striated. If the cysts die away, their contents change to a yellow, fatty, greasy and pulpy mass, which often seems to consist of chalk only. Pus may also be formed therein; blood rarely occurs. The whole can be distinguished microscopically only by the discovery of the characteristic hooks of scolices.

The echinococcus cysts grow in the lungs to a size varying from a pea to a human fist. There also they produce the same lumpy exterior and compression of the parenchyma, so that often a small part only of the original lung-tissue is left, while the compressed portions are extremely hard. Uhse compares such a lung to a "leather sack full of potatoes." The normal 6lb. ox-lung may weigh as much as 50lb. In one case Becker found 2,300 cysts in the lungs of an ox, and 5,000 in the entire carcase. When the cysts die out, a caseous, degenerated detritus is produced, resembling old nodules of tuberculosis.

In the heart only one such cyst is generally found, at the lower end of the septum, near the apex, and may enlarge either towards the ventricles or the pericardium. Echinococci occasion sometimes tumour-like swellings in the bones, *e.g.*, in the posterior maxilla; also in the medullary cavities of the tibia and the femur and they have been found in the sternum. They are, moreover, not uncommon in the muscles, particularly of pigs. Occasionally they occur in the udder.

Occurrence of Echinococcus.—Peiper estimates that echinococci occur with about equal frequency in the lungs and liver of cattle (8 : 7); in sheep, they are more usually met with in the lungs (3 : 2); in swine they rarely occur in the lungs (1 : 8). In man the echinococcus is found in the liver

in 60–70 per cent. of all cases. According to Widemeyer's analysis, echinococcus cysts contain: water, 90 per cent.; albumen, 6 per cent.; salts, 3 per cent.

Symptoms.—These vary according to the organs affected, but are not very characteristic. Cattle particularly may appear outwardly healthy, in spite of the presence of echinococci in large numbers. As many as 2,400 and 2,600 have been found in the livers of slaughtered cows, and 5,000 in the whole body, which showed no signs of the disease when alive (Becker, Posnjakow).

1. The chief signs by which they may be located in the *liver* are: disturbed digestion, gradual wasting and a general sickliness of some years duration. Jaundice is rare. The liver shows itself on percussion to be more or less enlarged on the right side in the region of the last two or three ribs, that is, from the tenth or ninth rib to the posterior edge of the last but one. Palpation proves its region to be sensitive. Sometimes a tumour can be seen in the neighbourhood of the right flank, or lumpy, fluctuating irregularities felt upon the liver, on examination through the rectum. Fever is usually absent, or occurs only with other complications, or in the last stages of the disease. When the superficial cysts burst, their contents pour into the abdominal cavity, where they may produce fatal peritonitis (rare).

2. The invasion of the *lungs* by echinococci causes (like tuberculosis) the aspect of pulmonary consumption, with gradually increasing emaciation, hardness of the skin, etc. The illness manifests itself in growing difficulty of breathing, which, when the invading echinococci are numerous, may develop with extraordinary rapidity, when respiration becomes a series of interrupted gasps, and a weak, harsh cough occasionally sets in. On percussion we obtain, according to the position of the cysts, a deadened valvular sound (rarely as though empty), the sound of the broken vessel (*olla rupta*), and an increased resistance under the hammer. On applying the ear or stethoscope, the normal vesicular breathing sound has ceased, and strange murmurs are heard—according to Harms, a squelching noise (the same sound that is heard on pressing or kneading a section of lung containing echinococci, and which Harms considers pathologically important). Also humming and cooing noises are perceived and (according to Schmidt) a peculiar metallic ring. On striking more forcibly

on the thoracic wall, the beast retreats and groans, particularly if struck in the region of the last four ribs. Fever is generally absent. The appetite and yield of milk may for a long time be fully maintained. If a cyst has penetrated the thoracic cavity, pneumo-thorax with fatal pleuritis may be developed.

3. Echinococcus cysts in the *heart* sometimes produce death resembling apoplexy by bursting or inducing paralysis of the organ. In such cases the animals drop suddenly and die, or exhibit symptoms of heart-weakness and suffocation.

4. In the *muscles* the cysts may cause lameness. Mégnin found the adductor muscles of the hind leg traversed by numerous cysts. Johne found one such in the psoas muscles of a horse. They are also found in the pleura and sub-cutis ; in the frontal cavity, and in the brain and cerebral membrane of the horse ; occasionally between the pleura and ribs, in the brain and in the eye of cattle (symptoms of staggers).

Prognosis.—Whenever a large number of cysts are present in any one organ, this is unfavourable, and death may result. Single echinococci may, however, become caseated or calcareous, and a great many are often found in the lungs and liver of otherwise healthy animals. A single echinococcus in the heart, however, may prove very dangerous. If once the disease can be identified, even though it be but approximately, the wisest course is to have the animal slaughtered.

Diagnosis and Differential Diagnosis.—This disease is by no means easy to identify. When in the *liver*, we must first of all demonstrate an enlarged liver-dulness, sensibility to pain in that region, dilatation of the organ, etc. Also we must discover whether the liver possesses a lumpy surface, by examining it through the rectum. These signs may, however, be confused with those of pregnancy, hydrometra, pyometra, new growth in the womb, ovarian tumours, and abdominal pregnancy, all of which are excluded by rectal exploration ; also with ascites, which may be known by percussion and by the changed conditions in the dorsal position ; and finally with other, and especially cancerous liver-tumours, which, however, are of rarer occurrence. There may also be uncertainty in diagnosing it as a case of hydro-nephrosis.

When the disease is in the *lungs*, it is chiefly important to differentiate it from tuberculosis. According to Harms, the following are the signs to note :—1. In echinococcus sickness

there is no cough, or it is very weak, whereas it is never absent in tuberculosis and is comparatively strong. 2. The squelching or croaking sound is in echinococcus sickness pathognostic. 3. Breathing is also more rapid and difficult in the last named sickness, and often, as compared with the animal's external condition (generally quite normal), this increased difficulty and rapidity of respiration is alarmingly sudden. 4. Friction fremitus is absent in hydatid disease, whereas in tuberculosis it is frequently (?) audible. 5. Of importance in echinococcus sickness is its complication with demonstrable enlargement of the liver. Moreover, in tuberculosis of the lungs we frequently find a co-affection of the lymph-glands as well as tubercular inflammation of the udder.

Treatment is of no avail. The utmost possible is, as in men, to attempt puncture of all accessible echinococcus cysts. On the other hand, prophylaxis is of the greatest importance. This consists in destroying the cysts, which ought to be legally obligatory, seeing their great danger to men and their economic injuriousness; also in ridding dogs of tænia and a reduction in the number of dogs by heavy taxation.

Echinococcus Sickness in Man.—This is by no means rare, and is of importance to veterinary surgeons because they are peculiarly subject to infection by tænia echinococcus when treating dogs for tapeworms. In Central Germany 5 per 1,000 deaths are traceable to this disease; and in Iceland from 5 to 10 per cent. of the population perish from it. The symptoms which develop slowly, consist in the formation of a fluctuating, soft, palpable and lumpy liver-tumour, with pressure and a feeling of tightness on the right side, dyspnœa and bronchial catarrh caused by pressure of the liver upon the diaphragm; atrophy of the liver, jaundice, with occasional formation of abscesses internally or externally. If located in the lungs, the cysts may (though only rarely) be coughed up, and the disease thus recognised. If in the kidneys, we find tumour there and pain round that region, and a few of the hooks in the urine. A cyst in the brain causes local symptoms. Death occurs, on an average, after five years. Peiper has shown that the frequency of this disease in man is proportionate to its occurrence in animals. In Mecklenburg and Pomerania it is prevalent.

III.—COCCIDIOSIS IN RABBITS. GREGARINOSIS.

Occurrence.—Coccidia, especially *coccidium oviforme* in the liver, that is in the bile-ducts and intestinal canal, occur enzoötically in summer in young rabbits. They have also been

found in the same organs of dogs, calves, pigs and sheep, in birds, amphibia, fish and in man. The disease seems to be connected with the food supply in certain districts. It is moreover transmitted to young rabbits by older ones suffering from liver-coccidiosis.

Natural History.—According to Pfeiffer coccidia belong to the group of *sporozoa*, which are monocellular, low protozoa, propagated by the formation of spores, and standing between the animal and vegetable kingdoms. Among these sporozoa three orders may be distinguished as important from the point of view of animal pathology:

1. Coccidia, whose chief form is *coccidium oviforme*. This latter lives in the epithelium of the bowels and liver (bile-ducts) of rabbits, where it produces so-called coccidiosis of the liver (formerly known as liver-gregarinosis of rabbits). It also causes enzoötic catarrh of the respiratory mucous membrane in rabbits (cp. Infectious Colds, Vol. II.). Coccidiosis of the bowels also occurs in birds as well as in mammals (cattle, dogs, swine, sheep), and is produced by a coccidium oviforme (cp. "Chicken Diphtheritis and Dysentery in Cattle," Vol. II.). Other kinds are *C. perforans and bigeminum.*

2. *Sporidia* (*psorospermia, sarcosporidia*) which live as parasites in the muscle cells of mammalia and birds, and also in the blood-cells (malaria, Texas fever).

3. The *Gregarines*, viz.: those living in swarms, very highly organised, naked sporozoa.

The *Coccidia oviforme* (egg-shaped), formerly designated gregarine or psorospermia, are, when fully developed, egg-shaped objects with a double contour, closely resembling oxyuris ova, and measure on an average $\frac{1}{700}$ by $\frac{1}{1500}$ inch. They settle in the epithelium of the mucous membrane of the bile-ducts and bowels, are enclosed in a firm, solid, transparent shell, and contain a coarse-grained protoplasmic substance. They appear to multiply wherever they are located, especially in the liver, by formation of spores, and also outside the body in the open. The contents of the coccidia break up into small, egg-shaped spores, which excrete a sickle or kidney-shaped transparent object, which strongly refracts the light. These are to be regarded as the young of the coccidia, which assume a globular shape without visible organization, and effect amœboid movement by extension or retraction of processes.

Post Mortem Conditions.—In gregarinosis the rabbit's liver is enlarged and sometimes has a lumpy surface. Its parenchyma is permeated by whitish-yellow nodules or cysts, in size from a millet-grain to a bean or hazel-nut, which sometimes lie so close together that the liver-tissue beneath is completely hidden. These nodules, often closely resembling tubercles or other new growths, are to be regarded as parenchymatous proliferations of the mucous membranes of the biliary ducts, and contain a thick, yellow liquid, in substance creamy, crumbling or caseous. Under the microscope they are seen to be composed of capsules, egg-shaped, coccidia, fatty-degenerated epithelial cells, loose nuclei, drops of fat, etc., and separated from the parenchyma of the liver by a thick capsule of connective tissue, with numerous nuclei and fibres concentrically arranged.

In the intestinal canal of young rabbits coccidia are found lodged in the epithelial cells; also in dogs, sheep, pigs, cats, poultry, etc. Sometimes they lie singly, at others in groups, when they form small, elevated, whitish spots on the intestinal membrane. Finally they may even cover larger areas of the mucous membrane in the form of pseudo-membrane. They are most numerous in the intestinal villi, which stand out as white spots; also in Lieberkühn's glands, whose surroundings sometimes show inflammatory infiltration with formation of ulcers. The presence of coccidia leads moreover to inflammation of the intestinal mucous membrane.

Symptoms.—In the case of young rabbits, sometimes an entire warren is swept away, and the symptoms consist in diminished appetite, yellow discolouration of the mucous membranes, discharge from the nose, weakness, wasting, exhausting diarrhœa, tympanites, staggering, etc. Death ensues with convulsions. In older rabbits no visible symptoms manifest themselves, in spite of the presence in the liver of considerable numbers of coccidia. In young pheasants, the symptoms are lassitude, backwardness in development, pronounced emaciation, weakness resembling paralysis, and diarrhœa. The birds usually die after a few weeks' illness (Ljöbring).

Differential Diagnosis.—Mistakes in diagnosis were formerly common. Hake, who first discovered the coccidia, thought they were pus corpuscles, and Lang took them for new growths. They are often confused with the larvæ of pentastomes, tapeworms and oxyurides (Willach). Concerning psorosper-

mious utricles, which in literature are usually treated with coccidia, we must refer our readers to the chapter on "Muscular Diseases."

Therapeutics.—In treating liver or bowel gregarinosis only prophylaxis is of any avail. Infection appears to reach the young animals from older ones who have already recovered from the disease. For this reason, segregation should be speedily resorted to, and stalls should be disinfected. Of internal remedies, at the utmost, a mercurial preparation, preferably calomel, might be tried as an anti-parasitic drug.

Other Parasites of the Liver.—Occasionally the livers of swine, sheep, lambs and calves are invaded by swarms of *cysticercus tenuicollis.* Such cases of *hepatitis cysticerosa* have been observed in swine by Dürbeck, and in goats by Hauptmann. Avéradère noticed an epizoötic cysticercosis in a herd of lambs. The symptoms were: Peritonitis; pleuritis; hundreds of cysticerces in the thorax, abdomen, liver and lungs; death occurred in 5–6 days. *Cysticercus pisiformis* is occasionally found in the liver. Bondeaud and Borgert discovered them in large numbers in the liver and lungs of two swine. Wolffhügel states that the tape-worm stilesia hepatica frequently occurs in the gall-ducts of sheep and goats in East Africa. Megnin observed a case of chronic hepatitis in horses, caused by *sclerostomum armatum.* Liver complaint is said by Dinviddie to occur in Arkansas as the result of stephanurus dentatus. The Bilharzia crassa, as it has been named by Sousino, is said to occur in the liver in 75 per cent. of all sheep in Sicily. It is frequently found in the gall-ducts of cattle in Sardinia, accompanied by distoma. Bedel found *heteracis maculosa* in the liver of pigeons, numbers of the birds dying in a state of great emaciation. Bilharzia was observed in the liver of a calf which died of rinderpest in Cochin-China. Smith and Lucet have described an inflammation of the lungs and liver accompanied by peritonitis in turkeys, which they attribute to a new protozoa (*Amœba meleagridis*).

Renal Coccidiosis.—This was observed by Railliet and Lucet in a goose (*coccidium truncatum*). There were numerous cysts in the kidneys the size of a pin's head and free coccidies were present in the uriniferous tubules.

CHAPTER V.

DISEASES OF THE PERITONEUM.

ACUTE PERITONITIS.

Classification.—Peritonitis may be classified from different points of view. It is first of all advisable to divide it into the acute and chronic forms, and this on purely practical grounds and in view of the course of the disease. Further, according to its distribution, we might define a circumscribed and a diffuse form; or to its origin, a primary or idiopathic, a secondary and a traumatic; we might speak of perforative peritonitis, tuberculous, sarcomatous, carcinomatous, pyæmic, metastatic, septic and rheumatic peritonitis. According to the nature of the products of inflammation, we distinguish a dry and exudative, a serous, sero-fibrinous, ulcerative, hæmorrhagic and an ichorous peritonitis. Regarding its origin, we generally have to deal with a secondary inflammation of the peritoneum, caused either by injury or by pathological processes in the adjoining parts. The possibility of idiopathic peritonitis, resulting from previous chill (rheumatic), cannot be disputed, though such cases are rare, for although rheumatic colic is not uncommon in horses, yet rheumatic peritonitis occurs only in isolated instances.

Occurrence.—Acute peritonitis appears in all domestic animals, including poultry. Females are, however, more predisposed to it than males, which may be explained by the closer connection existing between their organs of generation and the peritoneum. Thus, in cattle, it is far more common than in horses, because they are more frequently pregnant and are thus exposed to inflammation, rupture, etc., of the uterus during the act of delivery, from which peritonitis usually results. Moreover, in cattle, peritonitis not unfrequently follows as a complication of distomatosis of the liver, peritoneal tuberculosis, etc. The peritoneum of domestic animals differs singularly in

sensitiveness. That of the horse seems to be most sensitive, then follow the ruminants (sheep, goats, cattle), next the carnivora (dogs and cats), and last of all the omnivora (pigs) and poultry.

Etiology.—The causes of acute peritonitis are as follows: —1. Penetrating abdominal wounds, both accidental and such as result from operations. Under the latter head come all those caused by opening the abdominal cavity (laparotomy), such as herniotomy, puncture of the bowels or abdominal cavity, opening of the paunch (enterotomy), castration of the female animal and castration of subjects with undescended testes. The true cause of the inflammation is to be sought in the introduction of irritant substances (bacteria) by means of the hand or by insufficiently disinfected instruments. In rare cases infection is conveyed by the air which reaches the wound.

2. Rupture and perforation of organs covered by the peritoneum, with consequent intrusion of irritant matter into the abdominal cavity. Among these are ruptures of the stomach, bowels, urinary bladder, liver, spleen, etc.; laceration and perforation of the walls of the stomach or bowels by ulcers, foreign bodies, tightly-wedged fæces, and intestinal worms (gastrus larvæ, ascarides); rupture of the womb during parturition; rupture of the gall-bladder; perforation of the liver-capsule by distoma; penetration by abscesses, echinococcus-cysts; rupture caused by thrombosis of the vessels in hæmorrhagically-congested and narcotic bowels; perforation of the rectum during examination.

3. The spread of inflammation from an adjacent organ to the peritoneum, as, *e.g.*, from the abdominal walls after contusion; from the stomach or bowels after intense inflammation of their mucous membranes; from ulcers, partial ruptures, displacement of intestines (twisting, invagination, incarceration, strangulated hernia); further from an inflamed condition of the liver, kidneys, bladder, womb (metritis, para and peri-metritis), of the ovaries, testicles or of the stump of the spermatic cord; and finally, though less frequently, as a result of previous pleuritis.

4. Chills, whether external of the abdomen, or internal by absorption of cold drinks. According to Trasbôt, it may also arise from cold injections into the rectum. This so-called rheumatic peritonitis seems, however, to be rare.

5. On the occurrence of metastatic peritonitis in the course of pyæmia and septicæmia in our domestic animals little is as yet known. In many of the cases, especially among cattle, the cause remains obscure. It may result from lymphogenic infection, conveyed to the peritoneum from the intestinal tract (Bongartz).

Bacteriology.—Investigations have, in late years, been made into the micro-organisms which produce peritonitis. Predöhl assigns an important rôle to *streptococcus pyogenes* in the production of peritonitis, particularly of the traumatic form. Bumm differentiates between a streptococci-peritonitis (pus) and a putrid form (ichor), as well as a combination of both. The *bacterium coli commune* seems to be pathogenically important in perforative peritonitis (Ziegler, Jensen). Death seems in this case to result less from the peritonitis, *i.e.*, from its resultant septicæmia, than from poisoning occasioned by absorption of poisonous toxines from the fæces, and perhaps partly from nervous shock. In the serous exudation from a horse, Hamburger found a specific streptococcus, which he calls *streptococcus peritonitidis equi*. Boccalari found a diplococcus in the suppurative fibrous peritonitis of suckling calves. In a case of multiple suppurative peritonitis in cattle, Glaze observed the *bacillus pyogenes* (Grips).

Post-Mortem Conditions.—In acute diffuse peritonitis the peritoneum is found at the beginning of the inflammation to be hyperæmic and infiltrated by numerous minute dots of extravasated blood. The redness is of varying degree, sometimes uniform and at others irregular; its tint ranging from pink to brick-red and scarlet, at times streaky or spotted. The inflammatory injection is usually not equally advanced in all parts, and its starting point can often be traced. In order to see the hyperæmia of the peritoneum it may sometimes be necessary to wipe away the exuded matter lying over it. The redness diminishes later, and the peritoneum appears a greyish red. The upper surface of the serosa soon loses its lustre, becomes less transparent, raised like velvet, and the peritoneum itself easier to tear, and detachable. Newly-formed vessels quickly appear in the inflamed tissue. On the inner surface of the peritoneum an exudation, which varies much in form and quality, is deposited usually soon after the commencement of the disease. This sometimes consists only of a thin layer of coagulated fibrin in the form of a fine, translucent and easily removable membrane. In other cases this deposit is stronger, resembling a yellowish white or deep yellow croup-membrane. By means of this overlying exudation a part of the visceral layer often becomes attached to the parietal layer of the peri-

toneum ; or different organs in the abdominal cavity may be fastened to each other by their visceral peritoneal covering (*peritonitis fibrinosa* or *sicca*). In contrast to these dryer products of inflammation, a varying amount of yellow to yellowish-red fluid collects in the abdominal cavity in serous or sero-fibrinous peritonitis. This is at times greenish yellow, very turbid, and may, in horses and cattle, amount to eight and a half gallons ; it consists chiefly of serum, flakes of pus, fibrin, coagulated and cellular matter. If such exudations contain a large amount of red blood-corpuscles, the fact is shown by a brighter red colour, as has been noted especially in horses (complication of sero-fibrinous with hæmorrhagic peritonitis). In cases where there is a perforative peritonitis proceeding from the intestinal canal, this exudation is ichorous in appearance, very foul-smelling (ichorous peritonitis), and is usually mixed with extravasated contents of the bowel. After rupture of the bladder it has a strong smell of urine. In cats and dogs the exudation in the abdominal cavity is more purulent in character (*peritonitis purulenta*), and either rests on the peritoneum as a thin, creamy layer, or gathers in the abdominal cavity as a dirty yellow fluid, usually mixed with fine flakes and smelling offensively, especially after the breaking through of abscesses.

In the later progress of the peritonitis the exudations named are partially or entirely re-absorbed. To this process, the cellular elements and fibrin offer most resistance, as they must first undergo fatty degeneration and become crumbled into fine detritus. After resorption of the fibrinous exudations there often remain adhesions of the peritoneal layers (peritonitis adhæsiva), forming cords or bands of connective tissue which by the contraction of cicatrisation, may cause stretching and shrinking of the organs of the abdominal cavity, as well as constriction of the canals (bowels, biliary ducts), etc. If the resorption of the fluid exudations be only partially completed, and a permanent issue of white blood-corpuscles take place, the fluid obtains a purulent and at times a mucous-purulent appearance. In rare cases, if there be partial adhesion simultaneously with a discharge of small particles of exuded matter, these become with the progress of resorption more and more thickened, and transformed into a caseous and even calcareous mass. If the peritonitis last long, the intestine also shares in the process, in so far that, by the occurrence of a collateral inflammatory œdema, its walls are thickened, rendered moist and may become very opaque, resulting in paralysis of the muscular

apparatus of the bowel, and of the entire bowel-section, with stoppage, accumulation of gas, etc. The mucous membrane of the intestinal canal is also catarrhally inflamed. Large quantities of peritonitic exudate may produce injury by purely mechanical pressure upon the diaphragm and other organs of the abdominal cavity, as well as softening of the outer surfaces of such organs. In particular, infiltration of the diaphragm soon produces paralysis of this muscle and causes extreme difficulty of respiration (*dyspnœa*).

Acute circumscribed, or local peritonitis shows in general the same pathological changes, but they are restricted to smaller portions of the serosa, *e.g.*, to the serous covering of an intestinal part, of the liver, womb or ovaries. Such a peritonitis follows on wounds in the peritoneum (puncture), or on inflammation of any of the organs covered by it (*peri-metritis, peri-hepatitis*). The chief point of importance lies in a local restriction of the irritant matter by early cohesion of the inflamed layers or discharge of the exuded matter. Slighter cases of circumscribed fibrinous peritonitis often occur in our domestic animals, especially horses, as is proved by the traces of old disease found on dissection.

Phenomena of Acute Diffuse Peritonitis, with especial regard to Horses.—These can in no wise be termed typical or uniform, from the fact that peritonitis is, except in rare cases, a secondary complaint, and its symptoms may be changed in many ways by the primary disease, or even completely obscured. When the occurrence of peritonitis can be positively established during the course of the primary ailment, it is difficult, if not impossible, to separate the conditions of the two diseases during their further progress; for the primary disease may run its course with such terrible acuteness, that the symptoms of peritonitis may never come to light, as *e.g.*, in displacement of the intestines in horses. Moreover, the course varies greatly in the different kinds of peritonitis. Sometimes it is slow and continuous, and at others the symptoms, from the beginning, are severe and violent, and progress so rapidly that no special feature can be singled out. Thus in perforative peritonitis death occurs in a relatively brief time, and yet only the symptoms of sepsis can be identified. The most important signs, and those which may be regarded as especially belonging to peritonitis, are the following :—

1. The first sign is *acute pain* shown mostly by slight or

severe attacks of colic, which is often most violent at first and abates later, or shows remission. Thus in a case of traumatic peritonitis, proceeding from the spermatic cord, we have seen the pains appear quickly and very violently. They manifested themselves in grinding of the teeth, gnawing, snorting, blowing, plunging the head into the manger, spasmodic stretching of the extremities, rolling half-over on to the back, and then avoidance of all physical exertion, these symptoms being accompanied by partial and then profuse sweating. The pains either decreased during further progress of the disease, or became worse again after temporary abatement. In other cases the phenomena of restlessness are from the commencement to the end only very slight, and are restricted to frequent lying down and groaning, or even at times to mere pawing of the ground, looking backwards, drawing in the back, kicking at the abdomen and lashing the tail. In a few cases there seem to be no indications of pain whatsoever.

2. The *fever* is generally intense. In most cases of diffuse peritonitis we have noticed temperatures of from 41 to 42° C. (105.8 to 107° Fahr.). With perforative peritonitis the fever may increase very rapidly, accompanied by shiverings. The temperature is moreover irregular, as is also the course of the disease, and it is impossible to establish any definite fever-type. Its course is usually intermittent or remittent. In severe septic forms of peritonitis the temperature may even be subnormal. The pulse is almost always distinguished by an extraordinary acceleration out of all proportion to the bodily temperature, its speed with horses being often 80 beats and more per minute, rising even to 130. Characteristic of peritonitis is also the fact that at the beginning the pulse is, as a rule, feeble and hard, growing weaker with the progress of the disease, until it becomes quite imperceptible. The artery is very much contracted and somewhat resembles a wire. As the pulse grows more rapid and feeble, the palpitation of the heart increases. The visible mucous membranes, especially of the head sometimes become more highly reddened or cyanotic, and again grow pale. The superficial temperature is irregular, and the extremities in particular are cold.

3. The *abdomen* is at first very much drawn up, and more or less *sensitive to pressure*. In mild cases the pain is at first localised, whereas later it is more widespread as the process becomes diffuse. In severer attacks this pain disappears with the progress of the disease. Sometimes no indication of pain can

be found on pressing the abdomen, which may be explained by the much greater difficulty of applying the test of palpation to a horse, owing to its larger size as compared with other animals, and by the fact that it is not easy to examine this part in horses which are usually sensitive and ticklish. The abdomen also often becomes visibly enlarged early in the disease. The flanks are first distended by accumulations of gas in the intestines, and the tympany may become severe. A distension of the abdomen downwards and sideways is also noticeable in the smaller domestic animals (less so in the larger ones), caused by accumulation of exudate, which fact enables us to detect, both by palpation and auscultation, the same symptoms as we shall describe in connection with ascites. The peristaltic action of the bowels is completely suspended, often even in the earlier stages of the disease, but especially in its later course (paralysis of the bowels by œdematous saturation of the muscular apparatus). In consequence of this, defecation is delayed or entirely stopped, though the animal will often strain hard to evacuate. The dung is generally in small balls, dry and coated with a crust of mucus, and readily collects in great masses in the rectum ; yet severe diarrhœa may set in towards the end. We often observe difficulty in urination, which is shown by the animals frequently placing themselves in position to micturate. In prolonged pericystitis the urine may even be retained for several days (paresis of the sphincter). The passage of water, as well as of fæces, is rendered difficult, owing to the pain caused by contraction of the abdominal walls. As a rule very little urine is passed ; and in proportion to the time that the animals have fasted, it is generally acid, of high specific gravity and usually contains albumen. In one case we found, shortly before death, a great quantity of bacteria in the urine.

4. *Food consumption* may, according to circumstances, entirely cease, or gradually diminish. Later, it usually ceases entirely, as well as that of water. As in man, so also in the horse, eructation and vomiting are noticed, though rarely.

5. *Breathing* is usually accelerated, now short and superficial, then very deep and laboured, arising not unfrequently to a degree of intense dyspnœa. The causes of this dyspnœa are to be found in a paresis of the diaphragm, consequent on sympathy with its peritoneal covering ; in restriction of space in the abdominal cavity produced by the exudations therein and by the tympanically dilatated intestines ; in the painful contraction of the diaphragm and abdominal walls ; and in

disturbances of circulation connected with lowered activity of the heart. The respiration varies from 20 to 70, and even more, per minute; and this action is often entirely thoracic, owing to the paresis of the diaphragm and abdominal walls (remarkable rise and fall of the ribs, and striking participation of the intra-costal muscles). In cases where the diaphragm is affected, singultus is stated to have been observed with gaspings which shook the whole body; moreover, on palpation, great sensitiveness was found along the insertion of the diaphragm.

6. The *general condition* is, as a rule, greatly embarrassed. After disappearance of the colic attacks, extreme lassitude and sleepiness are shown, combined with debility. The animal stands with hanging head, or leans against the wall (one that we observed remained standing until death), takes no notice of its surroundings, shows great weakness, tremblings, local muscular twitching, a stiff and constrained posture, is difficult to move and sometimes neighs for hours before the fatal end. When once the animal falls, death, as a rule, quickly occurs.

Course.—The course of acute peritonitis is in most cases unfavourable. The causes of death are generally found to be sepsis, *i.e.*, poisoning, with great weakness and paralysis of the heart; also at times in suffocation. Death, especially in cases of perforative peritonitis, soon occurs, sometimes in twelve hours, and may on an average be expected within from 20 to 30 hours. In milder cases the disease may last from eight to fourteen days, until death. It may terminate in rare cases in recovery or may become chronic.

Acute Peritonitis in Cattle.—All reports agree that in cattle, peritonitis presents symptoms of so indefinite a nature that they are often difficult to recognize. Above all, the accumulation of exudations in the abdomen can only in exceptional cases be physically proved, and palpation rarely reveals signs of pain. Hence, the pulse, which in horses, is so helpful an indication, is in cattle hardly any guide, being under normal conditions irregular. The symptom of tympanites is unimportant, as it occurs frequently in other cases. In general, the appearances which may be noted are: an all-round increase of circumference of the belly, dull manifestations of pain, groaning, sensibility to pressure upon the walls of the abdomen, slight trembling of the abdominal walls; varying fever; ague; coldness of the skin; severe indigestion; constipation and tympanites, peculiar rumbling noises after each movement of the rumen (Otto); constrained movement; stiff postures; speedy collapse; inefficacy of the strongest purgatives; frequent, small or even impalpable pulse. The duration runs from a few days to a few weeks, and the end in about half the cases is fatal.

Acute Peritonitis in Dogs.—In dogs and smaller animals the disease is easier to diagnose, because they can be more thoroughly examined by palpation. It is sometimes marked by signs of pain, sensibility of the abdomen to pressure, clearly recognisable exudations in the abdominal cavity, constipation, tympany, stiffness of the hind limbs, groaning during evacuation, dyspnœa and fever. In *Cats* excessive tympanites is occasionally noticed.

Acute Peritonitis in Poultry.—This arises from perforation of the stomach or bowels by foreign bodies, from rupture of the Fallopian tube, parasites in the abdominal cavity (echinorrhynchus, mites, etc.), operations (castration), etc. The symptoms are prostration, fever, loss of appetite and groaning when the stomach is pressed, or during evacuation. Liginères and Petit observed an enzoötic occurrence of peritonitis in turkeys caused by *aspergillus fumigatus* (mouldy buckwheat). The peritoneum was either thickly spotted with light-green patches of mildew, or covered with a cloudy liquid containing spores.

Diagnosis.—As will be seen from the foregoing, diagnosis of acute peritonitis is very difficult, especially in the larger animals. But if the existence of pain in the abdomen, with accumulation of exudations therein, can be proved, then diagnosis is certain. The latter symptom can only protect us against confusing it with enteritis and colic. Another characteristic sign is the very small wiry pulse. Examination of breathing and pulse are of little value in preventing the disease being mistaken for pnuemonia, pleuritis or endocarditis. With reference to confusion of the peritonitic exudation with other conditions, compare the paragraph on the Differential Diagnosis of Ascites.

Therapeutics.—The treatment is both local, and internal. The inflammation of the peritoneum may be locally combatted by external application of cold bandages and, in smaller animals, by applying ice-bags to the stomach. The custom of rubbing in mercurial ointments cannot be universally recommended in domestic animals, owing to the danger of salivation. It can be used only on horses and dogs. On the other hand, turpentine and mustard oil may be rubbed in on ruminants. When the exuded matter accumulates excessively, its removal may be attempted by puncture. In ulcerative peritonitis, a wide opening of the abdominal cavity by laparotomy is often the only means of saving life. In such cases the cavity must be afterwards carefully disinfected and washed out with a weak solution of salicylic and boracic acids. Injections of Lugol's iodine solution are also made for the same purpose. Internal

treatment consisted formerly in administration of remedies to allay inflammation and of strong purgatives (tartar emetic, saltpetre, Glauber's salt). As, however, these remedies stimulate and increase peristalsis, practitioners are cautioned against their use, because, by spreading the peritonitic exudations over a wider surface, they also increase the irritation. We should prescribe calomel as a disinfectant for the bowels. In later years opium has been given to reduce the peristaltic action of the bowels, and this in large doses (horse 2½ drams, ox 4 drams, dog 1½ to 7½ grains of the powder). The increased constipation thus produced may be combatted by simultaneous lukewarm clysters and infusions. Where much fluid collects in the abdominal cavity, diuretics may be used (digitalis, squills, strophanthus, caffeine, juniper berries, oil of turpentine and acetate of potash), or such as stimulate the flow of saliva (pilocarpine, arecoline). The primary conditions which produced peritonitis should as far as possible be immediately dealt with (disinfection of the uterus in metritis, of the bladder, in cystitis; herniotomy in strangulated hernia, opening of abscesses, etc.). When the debility is very marked, or in order to combat fever, subcutaneous injections of camphor are suitable remedies.

CHRONIC PERITONITIS.

Etiology.—A diffuse chronic peritonitis is much rarer than the acute form, and occurs most frequently in cattle. It may develop from the acute stage, or arise independently. In cattle we have to consider the passage of foreign bodies from the second stomach towards the abdominal cavity; as well as injuries caused by these animals ramming each other with their horns; in dogs the punctures which are often made to relieve ascites; in horses, after puncturing the intestine, and in all animals chronic inflammatory processes in the organs covered by the peritoneum (womb, kidneys, liver). There are chronic tuberculous, sarcomatous and carcinomatous forms of peritonitis as part phenomena of tuberculosis (cattle and pigs), sarcomatosis (horses) and of generalised cancer. Parasites such as wandering strongyli, gastrus-larvæ and cysticercus tenuicollis appear to occasionally give rise to chronic peritonitis. According to Baldoni, the *bacillus coli* may be a cause of chronic peritonitis in the dog.

Post-Mortem Conditions.—The anatomical changes in chronic peritonitis are confined very often to limited areas

(perihepatitis, perimetritis), and consist chiefly in a gradually increasing inflammatory proliferation of the peritoneal connective tissue, which often leads to considerable thickening of the peritoneum, to the growth of regular capsules of connective tissue round certain organs, and, at times, to local fibrous, cartilaginous, or even bony areas, while the organs grow fast to each other, and to the peritoneum, thereby causing stretching, compression and shrinkage. In this deforming or distorting peritonitis, which may be best observed in pigs, the bowel-loops grow together into knotted convolutions, whose interstices are often filled with a purulent, gelatinous, oily exudation, which later becomes caseous or chalky. Chronic peritonitis with abundant fluid exudation is rare; but occurs, for instance, in the sarcomatous peritonitis of horses. Chronic hyperplastic peritonitis with multiple formation of cysts is rarely observed in cattle (Bertolini and de Benedictus).

Symptoms.—These are generally the same as in acute peritonitis, only much less severe. The disease is consequently also more difficult to demonstrate, unless it follows the acute form. Its course is very slow. In *cattle* the signs of colic and of pain on palpation are generally lacking, and often the only symptom left is the daily increase of circumference in the abdomen, and this in spite of lost appetite and continuous diarrhœa. To this an œdematous swelling of the lower abdominal region may be added later. The same signs are sometimes noticed in *horses*, in whom, besides the symptoms just named, intermittent, brief, attacks of colic may be noted, with great sensitiveness in the flanks, accompanied by a rapid but small pulse and medium fever. In *dogs* the differentiation between this disease and ascites is very difficult and often impossible.

Therapeutics.—In many points, the treatment corresponds with that of ascites. In both complaints, when there is a considerable quantity of exuded matter, diuretic and laxative remedies are to be used with puncture. In the beginning, and with dryer exudation, we recommend warm wraps in the form of Priessnitz's bandages, for the abdomen, with internal administration of iodine salts (iodide of potassium and of iron). Rinsing the abdominal cavity after puncture is also to be recommended with smaller animals. For this we generally use Lugol's solution diluted with sterilised water.

ABDOMINAL DROPSY. ASCITES.

General Remarks.—Ascites (dropsy of the abdomen) is an accumulation in the abdominal cavity of a non-inflammatory exudate, and occurs in all our domestic animals, including poultry. In a pronounced form it is most frequent in dogs, especially in very young or very old ones. In the Berlin dog-hospital, 318 cases of ascites were met among 70,000 dogs treated, or 0.5 per cent. of the whole. It is also not uncommon in sheep, cattle and goats. It is rare among horses; Woodger says that in 22 years he saw but one case. The reason probably lies in the greater activity and more regular feeding hours of horses, which militate against anything in the nature of stasis and its consequent transudation. Storch describes a case of ascites in swine.

Etiology and Pathogenesis.—Ascites is no uniform or independent disease, but rather a symptom of various other primary disorders. In this sense we may indicate its causes as follows :—

1. *Chronic affections of the heart, lungs, kidneys, portal veins, venæ cavæ, or mesenteric glands.* These have partly, general disturbances of circulation as a consequence (heart, lungs, kidneys); and partly, local disturbances (liver, portal vein, venæ cavæ, mesenteric glands); they cause circulatory disturbances, and especially venous engorgement with consequent transudation (ascites due to venous engorgement).

(*a*) Among *heart-diseases* we must mention : old-standing and not sufficiently compensated valvular defects; chronic pericarditis and epicarditis with enormous serous exudation, or adhesion of the pericardium to the heart (cor villosum); and hydropericardium.

(*b*) Of *lung-diseases* : Emphysema of the lungs, interstitial processes with consecutive sclerosis and atrophy (tuberculosis).

(*c*) Of *kidney-diseases* : Interstitial nephritis (Bright's disease); and degenerative process.

(*d*) Of *liver-diseases* : Above all interstitial hepatitis, which leads to cirrhosis of the liver (*e.g.* in distomatosis, hydatid disease); adenoma of the liver, etc.

(*e*) Among *diseases of the portal vein* : Compression by tumours, thrombosis, etc.

(*f*) *Diseases of the venæ cavæ:* Thrombosis of the posterior venæ cavæ (foreign bodies in cattle).

(*g*) Of *diseases of the mesenteric glands*: Caseous degeneration and atrophy, as, for instance, in the enteritis of foals.

In all of the above-named diseases the flow of blood from capillaries and veins (*i.e.* the flow of lymph) is hindered; engorgement, with increase of intravascular pressure, occurs, complicated with increased permeability of the vascular walls, all caused by disturbed nutrition of the vascular endothelium, resulting from engorgement. These are conditions which easily explain the extravasation of fluid-blood from the vessels. In cases of engorgement in the region of the portal vein and its branches, the capillaries and veins of the peritoneum, which form the commencement of that system, are especially affected.

2. *Hydræmia,* that is increase of the watery constituents of the blood, and a lessening of its solid contents, especially albumen—the so-called *hydræmic or cachectic ascites.* This hydræmia may appear either as a primary or a secondary condition, after severe, prolonged and very wasting sickness, thus especially in sheep and cattle (so-called dry rot, cachexia aquosa; worm-diseases, cachexia verminosa, etc.). Here the transudation is consequent upon an alteration in the walls of the vessels. With ascites, dropsy of the breast and pericardium and œdema of the skin are also present.

3. *Carcinomatous, sarcomatous and tuberculous new growths of the Peritoneum,* and healed inflammations of the same, are also considered to be causes of ascites. It would here be a question of exudative, rather than transudative processes, namely, of chronic peritonitis, although the occurrence of true abdominal dropsy on the basis of above-named changes cannot be denied. We must remember that ascites and chronic peritonitis are by no means easy to differentiate. In advanced stages of tuberculosis in old cows, we believe we have frequently discovered ascites caused by tubercular nodules, seeing that there were no inflammatory changes of the peritoneum. On the other hand, we failed to find any trace of ascites in a dog ten years old, which had carcinomata spread over almost all its organs, and whose peritoneum was most severely affected. According to Cadiot and Breton the chief cause of ascites in the dog is tuberculosis.

5. *There is no fever*, unless produced by complication of the primary disease. The *pulse* is usually accelerated, grows steadily smaller and is at last thready. The heart-beat is easily excited, and later becomes palpitating. The *mucous membranes* are pale; the conjunctiva in sheep is infiltrated with fluid and the skin also very white. The *coat* is lustreless and ruffled, and the extremities cold. Dogs often adopt a sitting posture with the fore feet widely stretched, and are very unwilling to move, are soon tired, and only lie down continuously when very exhausted, whereby decubitus is often developed, as well as involuntary urination, consequent on paresis of the sphincter vesicæ.

6. In later stages the *appetite gradually diminishes*, and also rumination and movement of the paunch. In dogs the appetite seems for a long time unaffected, but becomes fastidious, and they occasionally vomit. Defecation is alternately retarded and diarrhœic; sometimes tympany supervenes; urination, at first increased, later becomes diminished. The secretion of milk gradually diminishes, and by degrees the signs of general chronic disturbance of nutrition manifest themselves.

Course.—This is chronic, lasting months and even years. The bodily wasting continues, signs of anasarca appear on the hind parts, viz., on the prepuce, udder, hypogastrium and thighs, caused by pressure of the transuded matter upon the veins, and by general and gradually developing dropsy. The animals grow weaker and more emaciated, till at last they cannot get up, the eyes sink deep into their sockets, dyspnœa, and decubitus rapidly increase, profuse diarrhœa prevails (in cows sometimes retching and vomiting), and death finally occurs by paralysis of the heart or lungs or by general marasmus.

Diagnosis.—The diagnosis of ascites is made by inspection, palpation, percussion and auscultation of the abdomen. To ensure certainty, an exploratory puncture may be made in the case of the dog. The latter confirms the diagnosis even in cases of smaller accumulations of liquid, in which otherwise an opinion is always very hard to form, and with the larger animals almost impossible. Ascites is more easily recognised in the smaller animals, because examination can be more thorough.

More difficult than the demonstration of the presence of ascites is the discovery of its exciting cause, although both for therapeutics and prognosis, this is of primary import-

ance. General dropsy may be easily established as the cause of ascites, if there be a simultaneous dropsy of the skin (anasarca), subcutaneous cellular tissue, and diffuse dropsy of the cavities. It is more difficult to recognise specific heart or lung affections (see the respective chapters), and particularly interstitial chronic hepatitis, which is a frequent source of ascites, but difficult to diagnose, owing to the fact that shrinkage of the liver and other characteristic signs, such as icterus, cannot always be detected. Enlargement of the liver, accompanied by prominent changes of its surface, can be more certainly recognised by palpation as the cause of abdominal dropsy. The presence of biliary pigment in the urine is a symptom of no importance, as it often occurs in dogs with the slightest gastro-intestinal catarrh, and in severe degeneration of the liver (*e.g.*, from liver-flukes) such pigment is altogether absent. In dogs the search for an exciting cause is often rendered more difficult by a combination of two ailments, for instance, of a valvular defect with cirrhosis of the liver. Finally, new growths of the peritoneum may, in rare cases, be positively demonstrated by palpation.

Differential Diagnosis.—Of diseases which may be confounded with ascites we have to consider :—

1. *Peritonitis.*—This is accompanied by fever, great disturbance of general health, signs of pain, sensibility to palpation. Moreover, the specific weight of the peritonitic fluid, as obtained by puncture, is higher.

2. *Pregnancy.*—May be easily tested by manual investigation. The animal's general health is undisturbed, and the belly distended more on one side than the other.

3. *Obesity.*—Especially in older dogs, if combined with some valvular defect and much shortness of breath, this may lead to mistake. Differentiation must then be based upon auscultation and experimental puncture (inspection and palpation are not sure guides).

4. *Hydrometra, pyometra, dropsy of the membranes of the fœtus* (especially in cows), *cystic degeneration of the ovaries, abdominal cysts,* especially *abnormally large tumours* in the abdominal cavity (Cunningham found in a two-year-old foal a new growth weighing 236 lbs.) and *cystic kidneys.* All these may be recognised, by examination through the rectum or vagina, by exploratory-puncture, and by the stability of the various regions as proved by palpation and percussion. Whereas

in ascites the results of percussion vary with the animal's position, with abdominal or ovarian cysts they remain constant. An abnormal accumulation of dung in the rectum may also lead to error.

5. *Paralysis of the bladder with excessive distension.* Here palpation reveals a spherical, fluctuating body in the region of the bladder; and percussion, that its position does not change. Disturbed urination, use of the catheter and an exploratory puncture may all assist in diagnosis.

6. *Rupture of the urinary bladder,* with accumulation of urine in the abdominal cavity. This occurs most frequently in oxen after lodgment of urinary calculi in the urethra, and may be diagnosed from the precedent colic, cessation of urinary discharge, empty bladder, urinous perspiration, fever and appearances of collapse.

Prognosis.—This is so far unfavourable, that those anatomical changes in the organs which are the true causes of ascites cannot, as a rule, be removed, and that alterations in the blood (hydræmia) have already taken place before the veterinary surgeon is called in. From our own experience we consider that the prognosis is relatively most favourable in the case of young dogs, and in a few such we have seen the disease spontaneously disappear. In older animals a temporary recovery only can be expected, seldom a permanent cure. The prognosis of chronic peritonitis, with which this complaint was often confounded, especially in horses, is more favourable.

Treatment.—We should primarily seek to remove the affections of heart, lungs or liver which lie at the root of the ascites. As these are mostly beyond the reach of medical art, the treatment must fall back on regulation of diet (albuminous food) and removal of the accumulated liquid. For these ends, administration of diuretics and purgatives is most usual; also sialagogues and diaphoretics should be tried. Of diuretic remedies we recommend digitalis in combination with juniper berries and acetate of potash, squills, diuretin and caffein are also good diuretics. Tincture of strophanthus and calomel are occasionally prescribed. Drastic remedies are inferior to diuretics, for, when used continuously, they cause weakness. For dogs, gamboge, croton oil and jalap have been mostly employed. For all domestic animals, pilocarpine and arecoline should be especially used to stimulate salivation (sialagogues):

They must be given in small doses—and very cautiously where there is heart affection :—viz., pilocarpine to horses subcutaneously, 3 grains ; cattle, 4½ grains ; dogs, ⅛th grain ; and arecoline to horses, 1 grain. The former practice of dosing dogs with sublimate is to be condemned, although it occasionally met with success.

Puncture for ascites is advisable, and indeed absolutely necessary, in all those cases in which the exuded matter by its mass and weight presses the diaphragm too far forward, thus producing dyspnœa and imminent danger of suffocation. In all others the operation is useless, for as stoppage still remains, the abdomen quickly refills, and repeated tapping of this accumulation draws off too much albumen from the animal. We must also point out that with older dogs the apparently safe operation of puncture may sometimes cause sudden death, when, for instance, the animals grow very excited and offer resistance (heart-shock). Also, if the ascitic fluid be allowed to run off too quickly, insensibility and paralysis of the brain may result. The best results may be expected from puncture in young dogs ; and we have often noticed a cure from an introduction of the trochar. The injection of Lugol's iodine solution into the abdominal cavity after puncture, which is recommended by many, is probably of benefit only in cases of chronic peritonitis and of ascites following on degeneration of the peritoneum.

CHAPTER VI.

DISEASES OF THE SPLEEN.

General Remarks.—Although, as will appear from the foregoing chapters, diseases of the spleen are not rare, they have in general but little interest for special pathology and therapeutics; for, with very few exceptions, they cannot be diagnosed during life, and are, therefore, only subjects for pathological anatomy, the text-books on which we recommend our readers to consult. Moreover, the pathological changes of the spleen are mostly secondary processes of infectious disease, anomalies in the blood and other ailments, which are elsewhere treated (pyæmia, anthrax, leucæmia). Finally, a great proportion of spleen-diseases belong to the sphere of surgery (traumatic affections of the spleen). In order to make this work as complete as possible, and to demonstrate the unimportance of these complaints, which are not even mentioned in many clinical text-books on human medicines, we will deal briefly with the following three most common diseases of the spleen:—

1. Hyperæmia of the spleen.
2. Inflammation of the spleen.
3. Rupture of the spleen.

Hyperæmia of the Spleen.—Active or congestive hyperæmia of the spleen occurs secondarily in most infectious general diseases, especially in all cases of septicæmia (anthrax, sepsis, pyæmia, the erysipelas of pigs, etc.). Primary congestion of the spleen is believed to arise from overloading the stomach, or from a too sudden change from meagre diet to full rations (acute plethora). Acute hyperæmia of the spleen may lead to rupture, inflammation and hyperplasia of the organ. It is clinically marked by enlargement and swelling, but as the latter cannot usually be confirmed by palpation and percussion, an acute congestion of the spleen cannot with certainty be diagnosed, but only suspected from the accompanying general disorder. A *passive* hyperæmia of the spleen (*chronic congestion*

of the spleen) arises in all disturbances ot circulation which hinder the discharge of the splenic-vein, also in all chronic affections of the liver, heart and lungs. If long persistent, it leads to induration of the connective tissue.

Inflammation of the Spleen.—Most cases, as a rule, result from traumatic injuries of the organ, or wounds inflicted from without or from within, pressure, blows, crushing, shock, etc. This inflammation is also an accompanying sign of pyæmia, and may, moreover, develop from an acute tumour of the spleen. We distinguish anatomically, a simple *Splenitis*, known by extreme swelling and enlargement of the spleen, grey colour of the pulp and cellular infiltration of the latter, and *Abscess*, or purulent inflammation of the spleen, which is usually of traumatic or pyæmic (metastatic) origin, and may appear either singly or in numbers. The localised abscesses of the spleen are either encapsuled, or break through into the abdominal cavity. Finally, a third form is identified as *Perisplenitis*, an inflammation of the serous covering of the spleen, which leads to thickening of the capsule and adhesion of the spleen to its adjacent organs.

As the *symptoms* of inflammation of the spleen are of very undecided nature, the ailment cannot in most cases be demonstrated during life. This is the more easily explained by the fact that very serious inflammatory changes in the spleen appear to produce no visible change in an animal's general condition. Thus Möbius found on slaughtering a cow, which was apparently thoroughly sound and well nourished, that the spleen was changed into an abscess holding 5½ pints of pus. If signs of illness be manifested, they are mostly of an uncertain character, consisting in gastric disturbance, great emaciation, colic and fever. An inflammation of the spleen can be only with certainty diagnosed, when enlargement of the organ and painfulness in that region can be proved by inspection, palpation or percussion. According to Notz, traumatic, hæmorrhagic, swelling of the spleen occurs in sucking calves when they have been trodden on or kicked by their mothers. They become suddenly ill, refuse all food and lie with half-shut, weeping eyes. Their pulse and breathing are accelerated, peristaltic action is suppressed, the abdomen is moderately full, and the ears cold. Death follows in from twelve to eighteen hours. Traumatic splenitis is relatively the easiest to recognise. The *treatment* of this form of inflammation is purely symptomatic.

Rupture of the Spleen.—This is generally of traumatic origin, but may occur as a consequence of congestion of the spleen or of splenitis. The pathological changes vary according to the position and size of the rupture. If superficial and small, and the capsule uninjured, a small hæmatoma arises in the form of a boil, which may heal by resorption and cicatrisation. In the same way, rents in the capsule may also heal, as has sometimes been noticed in horses on subsequent dissection. More serious hæmatomata of the spleen show their presence by signs of colic, lassitude, paleness of the mucous membrane and steady wasting. They are rarely proved by palpation and percussion. In extensive ruptures of the parenchyma of the spleen, *e.g.*, in great rents of the capsule, the animals reveal signs of internal hæmorrhage, anæmia of the mucous membranes, cold extremities, rapid failure of strength, fear, sweatings, imperceptible pulse, fall of bodily temperature and death in a few minutes or hours. Sometimes periodically recurrent hæmorrhage is noted, with alternate improvement and deterioration of the general health. The *treatment* consists in administration of styptic remedies (ergot, hydrastis, tannin, sugar of lead and preparations of iron).

Diseases of the Pancreas.—These have still less clinical interest than affections of the spleen. Mégnin and Nocard have described a case of pancreatitis in a horse. It displayed slight weariness, weakness, lessened appetite, constipation, emaciation and icterus. Autopsy revealed an extensive enlargement of the pancreatic duct, which was occluded by a plug of albumen, compression of the common biliary ducts by the enlarged pancreatic duct, induration of the tissues of the pancreas, with catarrh of the salivary ducts. Mettam observed acute hæmorrhagic pancreatitis in the dog, characterised by vomiting where death rapidly supervened. *Post-mortem* showed the pancreas enlarged to four times its normal size, soaked through with blood, and traversed by necrotic groups. New growths in the pancreas also possess an interest more anatomical than clinical. They generally take the form of melano-sarcoma in horses and of adenoma and sarcoma in dogs. A few cases have been described by Nocard, Käsewurm, and Friedberger. In cattle, salivary concretions occur in the pancreas similar to those in parotitis. The pancreatic duct and its larger ramifications are dilated like a string of beads, their walls thickened, and the expanded ducts filled with small whitish stones, partly angular and facetted or cylindrical, varying in size from a millet-seed to a hazel-nut, and consisting chiefly of carbonate of lime (Baer). Two cases of pancreatic hæmorrhage, with sudden collapse, were observed by Prettner in the dog. In a four-year-old horse, which became very emaciated, in spite of its being well-fed, and finally died with symptoms of petechial fever. Wheatley found that the pancreas weighed 17 lbs. 10 oz., and showed chronic interstitial inflammation.

Among the cattle of Japan, as well as among the cattle and buffaloes of Cochin-China, distomum pancreaticum occurs very often in the ductus Wirsungianus, without, however, giving rise to appreciable symptoms of illness (Janson and Railliet).

Parasites in the Abdominal Cavity.—1. In horses are found: *filaria papillosa, strongylus armatus and cysticercus fistularis.* 2. In ruminants, *cysticercus tenuicollis.* 3. In pigs, *cysticercus cellulosæ and tenuicollis, sclerostoma pinguicola.* 4. In carnivora, *echinococcus cysts.* 5. In rabbits, *cysticercus pisiformis.*

CHAPTER VII

DISEASES OF THE URINARY ORGANS.

INFLAMMATION OF THE KIDNEYS. NEPHRITIS.

Classification.—Regarded from an anatomical or etiological standpoint, nephritis may be classified under a large number of headings, none of which, however, are entirely suited to the purposes of general practice. According to the anatomical portion of the kidney first attacked, the condition is described as either *parenchymatous* (affection of the renal epithelium) or *interstitial* (inflammation of the vascular apparatus and of the interstitial connective tissue). Nephritis may be *acute* or *chronic* ; *diffuse* or *circumscribed* (disseminated). Distinct from true *nephritis*—that is, inflammation of the substance of the kidney—is *pyelonephritis*, which includes, in addition, inflammation of the renal pelvis. Regarded from the etiological standpoint, nephritis may be *infective*, *bacterial*, *mycotic*, *toxic*, *traumatic*, *rheumatic*, *metastatic*, or *embolic*. It may be a *primary*, or a *secondary* condition arising in the course of other diseases. Finally, in its pathogenic aspect, nephritis is either *hæmatogenic* (that is, caused by the presence in the blood of substances which set up inflammation) ; or *urogenic*, owing its origin to conditions in the bladder and renal pelvis.

From among these groups, the clinical headings which present themselves as most suited to the requirements of modern veterinary science, are as follows :—

1. *Acute nephritis*, formerly known as "acute Bright's disease." This may be of infectious, toxic, rheumatic or traumatic nature, and may occur as glomerulo-nephritis, or as acute diffuse nephritis.

2. *Chronic nephritis*, formerly called "chronic Bright's disease." Included under this head, are chronic parenchymatous and chronic interstitial nephritis, as well as the final stage of the latter, the so-called granular and atrophied kidney.

3. *Suppurative nephritis* following on suppurative metastasis.
4. *Pyelo-nephritis*, viz.: inflammation of the kidneys and pelvis of the kidney, which often occurs in cattle.

Chief Clinical Forms of Nephritis in Man.—In addition to suppurative and pyelo-nephritis, we also clinically distinguish three chief forms of inflammation of the kidneys, viz.:—

1. *Acute nephritis*, usually developed in connection with infectious disease, and ending, either in recovery or death, but seldom passing into the chronic interstitial form. Chief *symptoms*: oliguria, excessive albuminuria, great specific gravity of urine, hyaline casts, epithelia of the kidneys, white, and occasionally also red, blood-corpuscles in the urine.
2. *Chronic parenchymatous nephritis*, generally fatal, or leading to atrophy. The course is slow. Chief *symptoms*: moderate oliguria, intense albuminuria, higher specific gravity, casts, white blood-corpuscles, fatty-degenerated epithelia and waste substance; red blood-corpuscles rarely; dropsy. *Anatomically* characterised by kidney-enlargement (large, white kidney) and intense desquamation of the epithelium.
3. *Granular and atrophied kidney, induration or sclerosis of the kidney.* Develops slowly, sometimes from the second form. Chief *symptoms*: Polyuria, slight albuminuria, very low specific gravity, scanty formative elements in the urine. No dropsy, but, on the other hand, hypertrophy of the heart. *Anatomically* characterised by shrinking of the kidneys and proliferation of connective tissue.

Definition of Bright's Disease.—Under this name some authorities include all kidney diseases which are accompanied by albuminuria, and others restrict the term to true inflammation of the kidneys, including amyloid kidney; others again to primary interstitial nephritis. In view of such a divergence of opinions, it would be well to reject this old name of "Bright's Disease," and to substitute for it the anatomical nomenclature. In veterinary pathology it has caused great confusion, especially in cases of hæmoglobinuria of horses.

I.—ACUTE INFLAMMATION OF THE KIDNEYS. NEPHRITIS ACUTA.

(Parenchymatous, catarrhal and hæmorrhagic nephritis, glomerulo-nephritis, inflammatory œdema of the kidneys, opaque swelling of the kidneys.)

Etiology.—Acute nephritis occurs in all domestic animals from various causes, but more often in horses, cattle and dogs. First, it may develop from a simple hyperæmia of the kidneys, or it may result from traumatic causes, blows on the back, falls, over-exertion when pulling or too sudden reining-in when going at great speed. It is sometimes produced by chill, especially in horses. It is necessary to bear in mind the close connection between the skin and the kidneys, and the indirect action of any

suppression of the activity of the skin. Acute nephritis very often occurs during infectious diseases, almost all of which cause an infectious or toxic nephritis by reason of the excretion of bacterial poisons through the kidneys. In mild cases this shows itself as an opaque swelling and fatty degeneration of the epithelium of the kidney. Here we have usually to consider septic and pyæmic processes, such as puerperal septicæmia in cattle. Other infectious complaints, such as influenza, murrain, strangles, angina, erysipelas, anthrax, foot-and-mouth disease, distemper, bronchitis, etc., are also sometimes complicated with acute nephritis.

For glanderous and tuberculous nephritis, see the respective primary diseases concerned.

The pathology is the same in cases of nephritis produced by poisoning. For instance, those caused by meadow-saffron, cantharides, oil of turpentine, croton oil, male-fern, squills, etc., and especially many diseases in cattle which are termed "hæmaturia." Tar, carbolic acid, iodoform, and particularly the metallic poisons, such as phosphorus, arsenic, lead, mercury, etc., all have the same effect upon the kidneys. Also the fungi of mould, smut, blight or ergot (hyphomycetes, uridineæ and ustilagineæ) excite inflammation in the kidneys, as well as certain insects, *e.g.*, caterpillars and plant-lice on cabbages. Even too liberal a feed of raw potatoes and special kinds of cotton-seed meal, will produce this result. *Secondary* nephritis may originate from the spread of cystitis and pyelitis. Finally, of great practical importance is the nephritis which arises in the course of hæmoglobinæmia of horses, and is caused by hæmoglobinous congestion of the kidneys. It was formerly held to be the primary origin of the complaint just named.

Post-Mortem Conditions.—Acute nephritis occurs, according to Kitt, in various forms in our domestic animals.

1. *Nephritis parenchymatosa* is a joint designation for all those cases in which rough anatomical methods cannot determine whether we are dealing with an opaque swelling or with desquamation, necrosis of the epithelium, serous or sero-fibrinous exudation. In such instances the kidneys are not swollen, or only slightly, more easily detachable from the capsule, and on dissection, of a dull, washed-out colour, that is, grey or greyish-red, or spotted with yellowish brown. Their medullary substance is more or less hyperæmic, even stained dark red, the Malpighian corpuscles stand out prominently, while the body

of the kidneys is of normal or softened consistence. Under the microscope we find the capillaries and veins enlarged and fully charged with blood, there are fibrinous and granular exudations between the tubuli uriniferi, granular, hyaline and epithelial casts (cylinders), detached epithelial cells, fatty degeneration, opacity, swelling and formation of vacua.

2. *Nephritis parenchymatosa hæmorrhagica* is characterised, in addition to the signs already detailed, by many red, minute blood-points, giving the kidney an almost black-red external appearance, which on section is dull grey, spotted with red, and in its medullary substance a deep red (erysipelas of pigs).

3. *Nephritis acuta diffusa* is distinguished by excessive enlargement and increase of weight (twice or thrice the normal size), as well as by the soft, friable nature of the tissue. The capsule is very loose, the upper surface of a bloody and bright colour, with grey and yellowish-grey patches. The microscope reveals considerable enlargement of the capillaries with extravasation of blood into the tissue-clefts, leucocyte casts in the distended urinary ducts, as well as cellular infiltration between them. The glomeruli are often full of blood and covered with sanguinary extravasations.

Symptoms.—The most important, and sometimes the only symptoms, are in connection with the urine (*e.g.*, in the course of severe infectious disease). This is first exceedingly rich in albumen, much reduced in quantity, and in graver cases the discharge of urine is suspended for several days. Thus Funk noticed anuria in a cow lasting for five days, and Friedberger a similar case in a horse of seven days duration. The causes are stoppage of the urinary canals by exuded cylinders (casts), extreme inflammatory swelling of the tissue of the kidneys and consequent compression of the ducts, as well as a diminished secretion of water by the affected organs. The specific weight of the urine is always much increased, and the liquid is of thicker consistence, often slimy, turbid and discoloured. Sometimes this develops to hæmaturia.

The microscope reveals the various kinds of urinary casts in great quantity, some covered with cells and white and red blood-corpuscles, and further, according to the desquamative, interstitial or hæmorrhagic character of the complaint, we find numerous epithelial cells and white or red blood-corpuscles. The discharge of urine often occurs in drops only and with much pain and pressure (dysuria, strangury). On exploration

through the rectum, the bladder is always found empty. The region of the kidneys (loins) is painful under palpation, and the in-drawing of the back provoked by pressure upon the kidneys or lumbar region causes the animal great pain. The kidneys are found on examination (when possible) to be enlarged and sensitive to touch. Frequently, and especially at the commencement of the disease, intense paroxysms of pain, so-called renal colic, are manifested. The animal's back is stiffly bent, and its gait is strained and staggering. It rises with difficulty, and remains standing, usually with outstretched legs or feet gathered under its body. Sometimes also in walking the foot on the ailing side is dragged somewhat, or not advanced so far as the other. In male animals one testicle is often drawn up.

Appetite is suppressed from the first; and in dogs vomiting is sometimes noticed. Constipation generally sets in, to alternate later with diarrhœa. Bodily temperature is slightly or moderately raised, *i.e.*, so long as there are no uræmic attacks. The pulse, which at first is strong and hard, grows afterwards more rapid but weaker. Nothing is as yet known of hypertrophy of the heart occurring in domestic animals after an acute nephritis lasting several weeks; also the affections of the lungs noticed in men during the later course of this disease (bronchitis, pneumonia) have not yet been observed. On the other hand, hydræmic swelling of the subcutis develops in the domestic animals, viz., on the head, breast, belly, scrotum, joints and in the abdomen, as well as dropsy in the cavities of the body.

In severer cases of nephritis with fatal ending the symptoms of uræmia occur in addition to the above, viz., stupefaction, spasms and dyspnœa. Pflug found, during the final stages in a cow with bilateral nephritis, the most violent eclamptic attacks, with opisthotonus and (in the intervals) complete coma. We ourselves observed in a horse with bilateral acute diffuse nephritis, about twelve hours before death, considerable stupor, reeling, staggering, general sweating, vague muscular twitchings over the whole body, combined with extraordinary increase of bodily temperature (almost 108° F.).

Uræmia.—Except in the dog, cases of true uræmia are uncommon among our domestic animals. Dammann once saw in a sheep epileptiform spasms alternate with sopor. In dogs, vomiting, weakness resembling that of paralysis, coma and fall of bodily temperature are noted as uræmic symptoms connected with urinary calculi, stricture of the urethra and paralysis of the bladder. In a horse we observed pronounced epileptiform attacks. For the nature of uræmia we must refer our readers to the text-books of general pathology. It consists, in brief,

of an accumulation of urinary constitutents in the blood and tissues, first, the products of metabolism resulting from decomposition of its albumen (ptomaïnes, leucomaïnes and toxines), and then the urea and salts of potash. Traube's theory, according to which uræmia is essentially the result of an acute œdema of the brain, and also that of Frerich, which states the conversion of the urea into ammonium carbonate in the blood to be the true cause of uræmia, lack confirmation. The symptoms of uræmia in man are head-ache, somnolence, restlessness, delirium, nausea, vomiting, a feeling of anxiety, slight twitchings, as well as severe eclamptic fits, after which uræmic amaurosis or deafness frequently remains. Later on follow vomiting, sobbing, diarrhœa, excretion of urea on the skin producing itching, retardation of the pulse followed by quickening, a bodily temperature either abnormally high or abnormally low and dyspnœa.

Course and Prognosis.—Acute inflammation of the kidneys is usually decided within from eight to fourteen days, but death may ensue in a shorter time. As a precautionary measure it is better to regard the prognosis as unfavourable, for the cases of recovery and death are about equal. The appearance of the uræmic symptoms, however, is very unfavourable. Death usually results from œdema of the lungs or brain. Development into chronic nephritis is rare, but *Zimmermann* records a case in a dog.

Differential Diagnosis.—Acute nephritis is often mistaken for an inflammatory condition of some other abdominal organ, thus for peritonitis, colic, enteritis, cystitis or metritis, and is consequently overlooked. This can occur only when we omit to examine the urine, which will in all cases ensure a correct diagnosis of nephritis without much difficulty. Moreover, a careful local investigation of bladder, uterus, etc., should ensure a correct diagnosis.

Therapeutics.—The treatment of acute nephritis consists first in rest and avoidance of all irritating food, as well as of irritant medicines, such as the rubbing of turpentine or cantharides into the loins. For dogs, as for men, an exclusive milk diet is to be recommended. We must endeavour to relieve the kidneys by diaphoretics, laxatives, diuretics, and perhaps also by local derivatives in the region of the kidneys, of which the application of cold or moist or warm packs would be most suitable. Perspiration can be produced in the larger animals by diligent rubbing with straw, wrapping in warm clothes, the use of moist, warm (Priessnitz's) bandages about the rump; and a flow of saliva may be stimulated by subcutaneous injec-

tion of pilocarpine (2 grains for horses; 7 grains for cattle; $\frac{1}{16}$ to $\frac{1}{8}$ grain for dogs), as well as arecoline ($\frac{1}{12}$ grain for a horse). Smaller animals, such as dogs or pigs, may be profusely sweated by hot baths, the temperature of which is gradually raised to that of the body, or by hot cloths. In uræmia, blood-letting is recommended. Of purgatives the most drastic are the best, because of their intense power of driving out water (calomel, aloes, gamboge). But such as excite the kidneys (croton oil) must be avoided. The same applies to diuretics, of which the alkalies, particularly acetate of potash, should be used, and, if threatened by heart-failure, digitalis, but especially diuretin and caffeïn. Against eclamptic fits, bromide of potash, chloral hydrate and inhalation of chloroform must be tried. It was formerly the custom to give astringent and styptic remedies in acute nephritis—such as tannin, cinchona, uva ursi, and sugar of lead (antiseptic).

Hyperæmia of the Kidneys.—This is either a passing condition or altogether secondary, and consequently has rarely to be diagnosed as an independent ailment. We must differentiate between an active (congestive) and a passive (engorgement) hyperæmia. 1. The causes of the *active* form are usually those of acute nephritis, the first stage of which in many cases represents congestion of the kidneys, and is hard to distinguish from this disease. Thus it occurs, *e.g.*, after the action of acrid food-stuffs upon the kidneys, among which must be mentioned the various kinds of anemone, and adonis, the asclepiadeæ, also damp, mouldy oats, containing fungi, or hay in a similar condition. Active hyperæmia of the kidneys, with polyuria, its chief symptom, occurs sometimes from the same cause, and in spring especially is enzoötic. Bad or dirty drinking water from puddles and marshes may also produce congestion of the kidneys. Any form of plethora or excess of blood in the abdomen occasions more or less hyperæmia of the kidneys, which, under favouring circumstances may develop to hæmaturia. Finally, active hyperæmia of the kidneys is an accompaniment of many infectious diseases (influenza, murrain, rabies). 2. *Passive* hyperæmia of the kidneys develops itself when there is general engorgement of blood as a result of heart or lung affection (valvular defects, emphysema or thickening of the lungs, exudations of the pleura), as well as on engorgement of blood in the posterior vena cava from aneurisms, thromboses or tumours, or abnormal accumulation of food or gas in the intestinal canal, also after thrombosis or compression of renal veins and renal arteries (continued thrombus from aneurism of the anterior mesenteric artery or artery of the kidneys).

Dissection shows that in both forms of hyperæmia the kidneys are enlarged, swollen, softened and highly reddened. In the active form it is the surface vessels which are filled, and in the passive the veins of the medullary substance. In active hyperæmia especially, the kidneys are saturated with moisture and very soft (parenchymatous œdema); the

capsule is loose, and the upper surface of the organ sometimes spotted with blood in consequence of hæmorrhage. In prolonged passive hyperæmia of the kidneys the organ assumes a dark, bluish red colour and becomes hardened by proliferation of the interstitial tissue (cyanotic induration). The epithelium of the tubuli uriniferi also suffers disturbance of nutrition in fatty degeneration, and sometimes hæmorrhage. Finally, passive hyperæmia may develop into nephritis.

Symptoms.—In *active* hyperæmia of the kidneys we find polyuria, with pale, thin urine of low specific gravity. The quantity may, in a horse, rise from about 4½ quarts a day to 5½ gallons and more, and the specific weight may sink from the normal 1040 to 1001–5 (a form of diabetes insipidus). The animals also hold their bodies in a slightly curved attitude and walk stiffly. There is no fever. In *passive* hyperæmia of the kidneys the quantity of urine is, on the contrary, diminished, and albuminuria sets in (even hæmaturia); hyaline casts also occur sometimes in the urine. There is no fever, but a slight weakness in the hind legs. The course is chronic. In active hyperæmia it is advisable to drain off to the intestinal canal; in the passive form adopt the treatment prescribed for the primary ailment (digitalis), active exercise, etc.

II.—CHRONIC INFLAMMATION OF THE KIDNEYS. NEPHRITIS CHRONICA.

(*Nephritis indurativa, fibrosa, interstitialis, fibro-plastica, sclerosis of the kidneys, atrophied kidney, cicatrized kidney, granular kidney, white spotted kidney, granular atrophy of the kidneys.*)

Etiology.—Chronic inflammation of the kidneys either primarily assumes a creeping, chronic course, or it develops from acute nephritis. The true causes of chronic inflammation remain practically unknown. They must, however, be sought in the action of injurious irritants upon the tissues of the kidney. These irritants may be both parasitic and chemical in nature. The bacillus of glanders must rank first among the parasitical irritants. Chronic nephritis is sometimes caused by the intrusion into the blood of inflammatory bacteria and their elimination through the kidneys. Hereby both the bacteria and their products of decomposition may act as irritants upon the tissues of the kidney. This explains the frequent occurrence of this disease in cows, which, in consequence of the act of calving, are much exposed to the action of septic matter from the uterus.

Other channels of entrance for the inflammatory agents are (in horses) ulcers in the bowels and larynx, and chronic bronchial catarrh with formation of bronchi-ectases and emphysema. An embolic chronic nephritis sometimes occurs in horses in connection with an existing worm-aneurism of the anterior mesenteric artery and of the renal arteries with metastasis and formation of thrombi. Senile vascular changes (atheromatous degeneration) may also be connected with chronic inflammatory processes in the kidneys in older animals, as in man. For instance, in a case of double interstitial nephritis in a seven-year-old cow, the renal vessels had undergone complete atheromatous degeneration and the left renal artery had become completely ossified (Eppinger). Frequent chills may give rise to chronic nephritis.

The latter disease is very often complicated with chronic inflammation of the pelvis of the kidneys (pyelitis), especially in cattle. This chronic pyelo-nephritis of cattle, which is distinguished by a special irritant factor, will be treated in a later chapter.

Post-Mortem Conditions.—The various forms in which chronic nephritis manifests itself are, according to Kitt:

1. **Nephritis fibrosa multiplex,** also spoken of as *sclerosis maculata*, presents multiple deposits of connective tissue and contractions of the kidney, which are crater-shaped and occur mostly in cattle. On the upper cortical surface we see yellow, yellowish-white and greyish-white islands dotted about, which are slightly sunk or scabbed over in streaks, and which on section contract into stripes. The kidney has a firm consistence and lumpy surface. Newly formed connective tissue, cicatricial tissue and cellular foci of infiltration are shown by the microscope, and all indicate an inflammatory origin of the sclerosis (*sclerosis ex nephritide*), in contrast to the sclerosis embolica which arises after emboli (*nephritis fibrosa multiplex embolica*, scabbed kidney, *ren cicatricosus*).

2. **Nephritis fibrosa diffusa,** called also *sclerosis totalis* or *diffusa* in contrast to the first form, consists in a general and abundant interstitial proliferation of the connective tissue, which hardens the entire kidney, especially of cattle. The kidneys are abnormally large, extremely hard, white, yellowish-white or whitish-grey in colour and resemble a large lump of connective tissue without any lobular separation. It is difficult

to cut, creaks under the knife and is formed of fibrous tissue alternated with chalky layers. Microscopically examined, the kidney consists of cicatricial tissue, very fibrous and made up of spindle-shaped and round cells, instead of the parenchyma of the kidney, of which latter nothing is left but canal-like spaces and empty clefts or stratified chalky substance. Between these are crater-shaped cellular infiltrations.

3. **Nephritis granulosa,** which must be regarded as a chronic fibrous inflammation of the kidneys, with tendency to shrivelling (shrivelled kidney). The upper surface in this form is uneven, wrinkled, lumpy, rough, granular (nephritis granulosa, or granular atrophy of the kidneys), the consistence is firm, the colour, white or whitish-grey and the capsule hard to cut. The shrivelled kidney of pigs is the product of countless, closely-ranked inflammatory foci.

4. **Nephritis fibro-vesiculosa** is a complication of fibrous inflammation of the kidneys with cystic degeneration of the tubuli uriniferi, with formation of numerous small vesicles.

Nephritis fibro-plastica.—Kitt thus names a nephritis (also as nephritis maculosa alba, or white-spotted kidney of calves) frequently found in slaughtered calves and distinguished by whitish-grey, spotted, somewhat prominent multiple foci, which are round and semi-spherical, varying from a millet-seed to a pea in size, and are wedge-shaped on section. The microscope reveals in the white foci a plentiful accumulation of colourless cells, fibroblasts and connective-tissue cells. According to Kabitz, white spotted kidney is a bacterial nephritis, the result of umbilical infection. It may also emanate from toxins, or infection by the bowels. Vaerst believes the white spots to be embryonic renal tissue (blastema patches). In two fresh cases of multiple embolic nephritis Willach found that the inflammatory condition was caused by distoma (cercaries), and believes that they reached the kidneys from the bowel by way of the blood-stream.

Glomerulo-Nephritis.—This is an accompaniment of various nephrites, both the parenchymatous and the different chronic fibrous forms, and is characterised by a preponderating inflammation of the Malpighian corpuscles.

Nephritis mixta.—A combination of interstitial inflammation and degenerative change of the epithelium, with slow course. Thomassen states that chronic nephritis in the horse is generally complex, *i.e.*, both interstitial and parenchymatous.

Nephritis simplex.—Simple cellular infiltration in the interstitial connective tissue, in the form of opaque blotches and streaky discoloration of the cortex.

Nephritis urica.—The renal gout of fowls consists in a uric-acid incrustation. The upper surface of the kidney is sprinkled with white prominent dots, the kidneys are much enlarged, hard and their section-surface marbled in yellowish-brown and red-brown.

Symptoms.—Chronic inflammation of the kidneys usually develops gradually without any definite symptoms. The animals are therefore far advanced in disease before submitted for treatment. The first striking signs are also of very general nature, such as loss of appetite and weariness, for which a superficial examination can find no organic cause. A characteristic indication of the parenchymatous stage is first obtained by the appearance of dropsical indications, viz.: swellings in the lower parts of the legs, of the breast and of the belly. These signs suggest examination of the urine, which alone can afford a sure index to the nature of the disease. We find, then, the amount of the urine diminished, its specific gravity increased, the urine very rich in albumen and containing casts, epithelium, fatty granular cells, and sometimes also red blood-corpuscles. Further examination reveals a very hard and tense pulse, a strong heart-beat, which can often be felt on both sides, very loud cardiac sounds, as well as an occasionally marked cardiac dulness, which are all symptoms of hypertrophy of the heart. The temperature in the rectum is but slightly or, at most, moderately raised. Great thirst is generally manifest. If at this stage the intensity of the disease rapidly increases, the symptoms already described of uræmia appear, accompanied by extreme wasting and exhaustion, of which the animals quickly perish.

If from this parenchymatous stage chronic indurative (hardening) nephritis develop, the aspect of the disease changes entirely. The transition from the first form to the second is at first marked by a fall in the specific gravity of the urine, from 1025 to 1015 (in horses). In pronounced cases of atrophied kidneys the specific gravity is only 1001 to 1010; the urine is very thin and plentiful, contains but little albumen and few casts of epithelium. At the same time the œdema of the limbs disappears. This is explained by increased hypertrophy of the heart, which, by causing greater blood-pressure, promotes absorption of the collected transudation caused by stoppage, thereby raising the quantity of urine, while lowering its specific gravity and proportion of albumen. Thirst is very intense.

If compensation on the part of the heart be sufficient, this condition may last for years. Such compensatory hypertrophy

of the heart leads, however, sooner or later to gradual and increasing incapacity of that organ. The pulse grows feebler and irregular; and to complicate the growing vascular congestion in the body, bronchial catarrh sets in, with chronic catarrh of stomach and intestines, diffuse œdema, shortness of breath, palpitation, dizziness and even pneumonia, pleuritis, pericarditis, along with general hæmorrhage of the organs. The urine again diminishes, while its specific gravity and albuminous contents increase, and death finally ensues under the aspect of an acute or chronic uræmia.

Therapeutics.—The treatment of chronic nephritis is, in the main, the same as that of the acute form, but the prospects of cure are less favourable. We should improve the general condition by nourishment and removing the anæmia; also, in cases of heart failure, by increasing the blood-pressure. Administer therefore, first of all, cardiac tonics and diuretics, such as digitalis, diuretin and caffeïn, tincture of strophanthus and also calomel. Pilocarpine and arecoline are also to be used here if dropsy be present. As a resorbent for the hyperplastic tissue in the kidneys, iodide of potassium may be recommended.

Relation of Chronic Nephritis to Hypertrophy of the Heart.—There are various opinions on this subject. 1. According to Traube, the blood-pressure is raised by the accumulation of water in the blood and the disturbance of circulation consequent on changes in the liver, the result being a compensatory hypertrophy of the heart. 2. In opposition to this mechanical theory is Senator's chemical assumption, that the increased blood-pressure is caused by retention of the urinary constituents in the blood, and particularly of urea. In the absence of further data we can form no correct decision.

III.—PURULENT INFLAMMATION OF THE KIDNEYS. NEPHRITIS PURULENTA.

(Abscess of the kidneys, Embolic purulent Nephritis, pyonephrosis).

Etiology.—The pathogenic agents of purulent nephritis reach the renal tissues in two different ways, from the blood (hæmatogenous) or from the bladder by the ureters (urogenous). Purulent, ichorous, and, indeed, any infective emboli produce local inflammatory foci, as in septicæmia, endocarditis, gangrene of the lungs, pneumonia, pharyngitis, etc. Emboli of a non-

infective nature, such as those arising from worm-aneurism, produce an ordinary chronic but not purulent nephritis, with congestion and formation of new growth. Purulent nephritis, in the form of abscesses in the kidney, is most common in cattle. We have here to consider septic infection caused by hard labour-pains, or retention of the after-birth, with deposit of bacteria in the excretory organ of the kidneys. *From the bladder*, inflammation may spread by way of the ureter, first to the pelvis of the kidneys, and thence to the kidneys themselves (pyelo-nephritis). Finally, suppuration in the pelvis of the kidney may be caused by traumatic influences, for instance, by renal calculi. The paranephritic abscesses (psoatic abscesses) which occur, though rarely, in the vicinity of the kidneys in the horse, are usually abscesses sinking inwards, with fistulæ leading outwards to the inguinal or lumbar regions.

Post-Mortem Conditions.—According to Kitt, purulent nephritis occurs in domestic animals, chiefly in two forms.

1. *Diffuse Purulent Nephritis* arises when the pyogenic matter is distributed by the blood over the entire kidneys (nephritis purulenta punctata, diffusa, mixta). The whole caspule is permeated by numerous small abscesses in the shape of pus-coloured, whitish-yellow dots and streaks, which are surrounded by a bright red area and dark red hæmorrhages. The sectional surface of the kidney displays a bright appearance, sprinkled with white, grey, yellow, brown and red. These small abscesses in the cortex have their points towards the boundary and medullary layer, and contain numerous bacteria, especially clumps of micrococci. This disseminated form of purulent nephritis occurs chiefly among pigs and calves.

2. *Nephritis apostematosa, or abscess of the kidney*, is demonstrated either embolically, as in horses during the progress of strangles, or urogenously, as in cattle during purulent pyelo-nephritis. We find in the kidney purulent cavities, either singly or in numbers, from the size of a hen's egg to that of a man's head, with creamy pus and speckled-white borders, in which (in strangles) the presence of the streptococcus of strangles can be detected. In other cases the whole kidney is changed, after destruction of the parenchyma, into a fluctuating abscess, the walls of which consist of the capsule of the kidney and the indurated cortical substance, whereby the extended pelvis of the kidney is drawn into the abscess-cavity (pyo-nephrosis).

Symptoms.—These are not very characteristic, apart from the fact that they are masked by the symptoms of the primary disease (pyæmia, pyelitis, etc.). Often only great emaciation, disturbed appetite, increase of thirst, weariness, roughness of coat, etc., can be noticed. In other cases the urine offers some points for guidance, smells badly, is of thick, slimy consistence, mixed with pus and worm-shaped coagulæ, and blood and dead kidney-tissue may be seen in it. In cows the tail is found smeared with pus and mucus. In later stages embolic pneumonia, pyæmia, hæmorrhagic diathesis, etc., may occur. The course is generally slow and chronic, and the changes in the kidney are not known until after slaughter. Occasionally the presence of soft, fluctuating abscesses can be recognised in cattle by exploration of the abdominal cavity through the rectum.

Therapeutics.—Treatment is purely surgical, and may consist either in puncture of the pyogenic membrane or abscess from the lumbar region, or in extirpation by laparotomy. The latter operation was tried on a cow by Münich. Though in this case the result was not quite satisfactory, yet the operation may be recommended, seeing that cows are not highly sensitive to surgical treatment, and may be killed without much pecuniary loss should the operator prove unsuccessful. Maksutow claims to have successfully removed the kidney from a horse.

INFLAMMATION OF THE PELVIS OF THE KIDNEY. PYELITIS.

Etiology.—Inflammation of the pelvis of the kidneys usually results from the spread of inflammation from the bladder or kidney to the mucous membrane of the pelvis (pyelo-cystitis and pyelo-nephritis). It also often develops in the course of infectious diseases and of poisoning cases, when the infectious or poisonous matter comes in contact with the mucous membrane of the pelvis of the kidney on being excreted from the body. We may here remind our readers of glanders and tuberculosis, as well as of the action of acrid vegetable poisons. A special infectious form is the bacillary pyelo-nephritis of cattle which will be dealt with under a separate heading (page 340). Moreover, foreign bodies, especially renal calculi, cause inflammation—more or less severe—in the mucous membranes just named (pyelitis

calculosa). The presence of gravel or sand in the pelvis of the kidneys may also produce pyelitis, as well as that of the giant palisade worm (*eustrongylus gigas*) in dogs, horses and cattle. Accumulation of urine in the pelvis of the kidney, after mechanical or dynamic retention (stone in the bladder, or in the ureter), likewise produces pyelitis as a result of the decomposition of the urine. If such retention is of long duration, atrophy of the kidney may develop, with cystic breaking down of the renal substance (hydro-nephrosis).

Post-Mortem Conditions.—According to the degree, cause and duration of the inflammation, we may distinguish between simple, purulent, croupy, diphtheritic, calculous and also acute and chronic pyelitis. In the milder form the mucous membrane is swollen, reddened, sometimes permeated by hæmorrhage, as well as covered by a coating of mucus, rich in desquamated epithelial cells, which, in purulent pyelitis, consists almost entirely of white blood-corpuscles. The effect of renal calculi upon the mucous membrane manifests itself either by the occurrence of circumscribed, inflammatory foci, progressing sometimes to necrosis and formation of ulcers, and of croupous-diphtheritic deposits; or else it is shown in the form of an incrustation, with inflammation of the mucous membrane, in which the excreted sediment combines with the mucous membrane to form a rigid coating. Renal calculi in the horse occasionally attain a large size. Levin found one weighing 1 lb. 9 oz.; Bovet, 1 lb. 1½ oz.; Gräfe, 1 lb.; Falk found four stones with a combined weight of 2 lbs. 14 oz. These stones frequently give rise to *hydro-nephrosis* (cysto-nephrosis, cystic kidney). In cattle, urinary calculi have been found ranging in size from a pea to a pigeon's egg (Imminger). Chronic catarrh of the pelvis, so common in horses, is revealed in a dirty brownish discoloration of the kidney, with pigmentation, thickening, submucous infiltration and ulceration of the mucous membrane, accompanied by enlargement of the pelvis. Pyelitis soon becomes complicated with nephritis or cystitis, and the purulent form leads especially to purulent nephritis and pyo-nephrosis. Eustrongylus gigas is, in isolated cases, found to have wandered from the pelvis of the kidney into the abdominal cavity near the kidneys. Eustrongylus gigas, the giant palisade-worm, is a round-worm, varying from 4–28 inches in length, of about the thickness of a lead pencil, and blood-red in colour. It is generally found singly, coiled in the renal pelvis or in the renal

tissue, where it gives rise to severe hæmorrhage with cyst-like changes in the renal parenchyma. Occasionally, it gets into the urethra or into the bladder.

Symptoms.—The symptoms of this form of inflammation are very indefinite, owing to frequent complication with nephritis and cystitis. Pyelitis can only then be with certainty diagnosed when, on microscopic examination of the urine, the characteristic epithelial cylinders fitted with processes (shaped like molar teeth) are found, such as are peculiar to the lower layers of the mucous membrane of the pelvis. Sometimes in horses an enlargement of the pelvis, with thickening, *i.e.*, widening of the urinary channel may be demonstrated by palpation of the kidneys from the rectum and, in dogs, by palpation of the abdomen. In cattle, it is said that the presence of renal calculi may be detected by pressure upon the posterior lobe of the kidney, when distinct grating of the stones will be felt (Imminger). All other symptoms agree with those of the primary disorder. Renal calculi produces colic and difficulty of urination (so-called kidney-colic), and in long retention of urine may lead to uræmia. Eustrongylus gigas caused symptoms in two dogs which led to suspicion of rabies ; in others, it gave rise to dysuria and hæmaturia. In a few cases the worm has been known to find its way into the abdominal cavity, into the bladder, the urethra, the perineum, and the mammæ (Megnin). Rondelli and others have shown that *eustrongylus* may occur as an incidental condition in dogs with true rabies. For this reason, an inoculation of rabbits for purposes of diagnosis is indicated in all cases. In the urine we find, according to the type of pyelitis, pus corpuscles, coagulæ of blood and fibrin, necrotic mucous tissue, sand, and gravelly lumps and numerous strongylus ova, if eustrongylus gigas be present in the pelvis. Imminger says that where urinary calculi are found in cattle, the urine is as clear as water.

Therapeutics.—The treatment of pyelitis corresponds with that of the primary disease, and consists chiefly in administration of anti-catarrhal, antiseptic and astringent remedies, such as the alkalies, boracic acid, salicylic acid, creolin and all those medicines which contain tannic acid.

PYELO-NEPHRITIS IN CATTLE.

Etiology and Pathogenesis.—Among inflammatory disorders of the kidneys pyelo-nephritis occupies an independent position as it is caused by a special infectious agent, the bacillus renalis bovis (*bacillus pyelo-nephritidis bovis*). This bacillus, according to Enderlen and Höflich, who simultaneously discovered it, forms immovable, slender or slightly curved rods of 2–3 microns in length and 0.7 micron in breadth (a micron being $\frac{1}{25,400}$ of an inch), with rounded angles, which lie in heaps together and can easily be stained by Gram's method. The bacillus, which flourishes best at a temperature of 98.6° Fahr., forms on a firm nutrient ground, colonies which are grey, dotted, protuberant and very lustrous, and in bouillon a fine granular sediment. Sometimes the source of infection is complicated with pus-bacilli (streptococci, bacillus pyocyaneus).

Bacillary pyelo-nephritis is not a rare form of disease among horses, as observations made in the slaughter-houses have testified. The fact that it occurs mostly in mares and usually appears soon after parturition, points to some connection with septic processes in the uterus, and especially to retention of putrid after-birth. The intrusion of the bacillus from the womb to the kidneys may probably often occur by urogenous means, *i.e.*, through the vagina, urethra, bladder and ureter. The probability of such an ascensive inflammation is confirmed by the fact, that vaginitis, urethritis and cystitis are actually often present with pyelo-nephritis. The anatomical position of the orifice of the urethra on the under side of the vagina favours the access of micro-organisms to the bladder. On the other hand, bacillary pyelo-nephritis may be caused by hæmatogenous means, as experiments in intravenous transmission have shown that blood can convey the disease.

Non-bacillary Pyelo-nephritis.—In addition to the common form of pyelo-nephritis, which owes its origin to the *bacillus renalis bovis*, there is a form found among children which is not associated with any bacillus (Albrecht) or micro-organism. Liénaux and Zwaenepol found the specific bacillus in five cases out of six; Cadéac found staphylococcus pyogenes; Kitt found micrococci; Porcher, the bacillus coli; Moussu, different bacilli, bacilli coli and streptococci pyogenes.

Post-Mortem Conditions.—The bacillus of pyelo-nephritis causes pus formation and necrosis in the kidney

Around the inflammatory foci interstitial proliferation of connective tissue is produced in the later stages, with sclerosis. We may find one or both kidneys affected, and usually enlarged to double the normal size. The capsule of the kidney is often opaque and in places adherent. The upper surface of the kidney has a spotted appearance, in which clay-coloured or greyish-yellow circumscribed foci, from a pin-head to a pea in size, or even larger, alternate with dark red patches. Occasionally, too, this surface is lumpy and covered with numerous small abscesses. The substance of the kidney is harder than usual. On section we find yellowish-grey, yellowish-white or white streaks in the cortical substance, and at times also minute abscesses arranged in rows; similar light-grey stripes are also seen in the medullary substance. The renal papillæ are often covered with a dirty yellow purulent substance, or else are ulcerated and necrotic (necrotic papillary nephritis). The pelvis of the kidney is enlarged and contains dirty grey or yellowish-brown matter, which is purulent, mucous, flaky, crumbly or thinly fluid. Its mucous membrane is thickened, spotted with red, rough and occasionally covered with ulcers or warty proliferations. The urethra often displays similar changes and is usually from three to five times its normal thickness. Microscopical examination of the tissue of the kidney reveals minute cellular infiltration in the cortical substance, increase of interstitial tissue in cortex and medulla, atrophy of the urinary tubules, obliteration of the glomeruli, swelling and destruction of the epithelium of the tubuli uriniferi. In the pelvis of the kidney are numerous triple-phosphate (coffin lid) crystals, pus corpuscles, rod-shaped and flat epithelial cells and detritus, as well as yellowish-brown and greyish-brown collections of bacteria. At the lower end of the urinary tubules many bacteria may, by staining, also be discovered.

Symptoms.—As the course of pyelo-nephritis is chronic, its symptoms are uncertain. Animals manifest a gradually increased disturbance of general health, beginning with lessened appetite and rumination, and leading to great emaciation. This process is interrupted at times by intermittent colic and intermittent fever. Many cows stand with back arched and sensitive to pressure. Uræmic appearances, such as retention of urine, vomiting or colic, are rarer. The most important changes are to be found in the urine which, in advanced stages of the disease, is mucous, purulent and even bloody, and the surroundings of the pudenda, especially the tail, are often smeared in consequence

with putrid and bloody matter. Microscopic and chemical examination of the urine reveal the presence of white and red blood-corpuscles, rod-shaped and flat epithelial cells, triple-phosphate crystals, numerous bacilli and albumen. Examination of the kidneys through the rectum generally yields a negative result, though occasionally the enlarged right kidney and thickened urethra may be felt with the hand from the rectum (Hess, Sarsen, Imminger and Bartels). Sometimes the aspect of the disease is modified by complication with cystitis, metritis and vaginitis. As an indication of catarrh of the bladder we must notice: frequent urination, sometimes even strangury, pain during urination and constant desire for micturition. A simultaneous affection of the uterus and vagina reveals itself by a purulent, mucous or bloody discharge quite independent of urination.

Differential Diagnosis.—This chronic pyelo-nephritis can with certainty only be clinically distinguished from the chronic nephritis (atrophied kidney) already described when the presence of bacilli can be proved in the urine. This applies also to simple pyelitis, especially its calculous form. Without such proof pyelo-nephritis is difficult to distinguish from simple nephritis, because culculous pyelitis, by the spread of the inflammatory process from the renal pelvis to the renal parenchyma, may be complicated with chronic nephritis. The bulk of the cases of chronic nephritis in cattle are, however, really bacillary pyelo-nephritis, and, as a general rule, the clinical appearances offer sufficient ground for a diagnosis of its probable cause.

Therapeutics.—Treatment of bacillary pyelo-nephritis is usually of no avail, for which reason an early diagnosis is very expedient, in order that the owner may be advised to slaughter the animal before emaciation renders it worthless. If, however, an attempt at cure must be made, give internal antiseptics, to disinfect the diseased parenchyma of the kidney. Of such we would name: camphor, oil of turpentine, salicylic acid, chlorate of potash and folia uvæ ursi $\frac{2}{3}$ oz.–1$\frac{2}{3}$ oz. Camphor is usually given as an emulsion with linseed decoction; folia uvæ ursi as a decoction. In addition to these the remedies prescribed for nephritis may also be used.

Amyloid Kidney.—Amyloid degeneration of the kidneys has hitherto been rarely met with among our domestic animals as an inde-

pendent disease. It occurs more frequently with amyloid degeneration of other organs, and is more or less subordinate. Rabe noted it as an apparently independent ailment in three dogs, and Gerlach in an ox. The *anatomical changes* consist in a wax-like hardness of the cortical parenchyma, which is pale, dry and poor in blood, enlargement and glassy swelling of the glomeruli, together with the presence in the tortuous tubuli uriniferi of homogeneous, dully lustrous urinary casts, which slightly refract the light. Treated with methyl-violet, these parts take on an intense purple red, and with a watery solution of iodine in iodide of potassium a mahogany red, which last changes to steel-blue on the addition of sulphuric acid. According to Rabe's observations, the phenomena consist in dropsical swelling of the subcutis, especially of the limbs, in ascites, albuminuria and in uræmic symptoms, viz., vomiting, weakness like that of paralysis, coma and fall of bodily temperature. No hypertrophy of the heart was in any case noticed, but Dörflinger describes such a case in a dog. The *treatment* of amyloid kidney is the same as that of chronic nephritis, with which, during life, it is often confounded.

Tumours of the Kidney.—These are but rarely subjects of clinical investigation and treatment. In some cases, with small domestic animals, especially dogs and cats, these tumours (carcinomata, sarcomata, melanomata, adenomyomata, sarcomyomata and rhabdomyomata) may be clinically demonstrated by palpation of the abdomen. Generally, however, they are revealed only by dissection, when, during life, hæmaturia, sensitiveness in the renal region and disturbed urinary secretion have been observed. *Treatment* would merely consist in extirpation of the diseased kidney.

Coccidiosis of the Kidneys.—Railliet and Lucet noted a case of liver complaint produced by coccidia in the kidneys (compare p. 297).

RETENTION OF URINE—RETENTIO URINÆ.

General Remarks.—Retention of urine, also known as strangury, dysuria, and urinary colic, consists in an abnormal accumulation of urine in the bladder, with consequent distension and inflammation of the same, as well as of the ureters and pelvis of the kidneys, which leads eventually either to rupture of the bladder and peritonitis, or to atrophy of the kidney (due to pressure), to hydro-nephrosis and uræmia. It is not an independent disease, but merely a symptom of various maladies, and occurs most frequently in cattle, dogs, horses and sheep, chiefly in the male (*i.e.*, the castrated) animals, rarely among females.

Etiology.—The causes of this retention are as follows:—

1. *Foreign bodies in the bladder and urethra,* which impede the flow of urine. These include cystic and urethral calculi,

urinary sediment in the S-shaped bend of the urethra in oxen and wethers, broken pieces of catheter, or foreign substances purposely introduced into the urethra (straws, bits of broom), blood-coagulæ or particles of fibrin, portions of croupy and diphtheritic membrane in the bladder and polypoid tumours which cover up the orifice of the bladder.

2. *Compression with obstruction of the urethra and orifice of the bladder by adjacent new growths*, especially of the prostate. Then compression of the uterus, ovaries or of the bladder itself by accumulation of dung in the rectum, displacement of the uterus, pregnancy ; narrowing of the lumen of the urethra by stricture, inflammation, swelling or torsion of the penis, fracture of the os penis, paraphimosis (strangulation of the glans penis by swollen foreskin), phimosis (abnormal narrowing or obstruction of the orifice of the prepuce).

3. *Paralysis of the Urinary Bladder* (detrusor urinæ), in the form of a paresis or paralysis following upon severe croupous, diphtheritic, phlegmonous cystitis, pericystitis, affection of the spinal medulla (traumata, meningitis spinalis, apoplexy) or general weakness, after too prolonged a suppression of urination in horses suffering from brain affection, or which have been too long in movement, and lastly after reflex paresis of the muscular apparatus of the bladder during colic in the course of rheumatic hæmoglobinæmia of horses and in severe general disorders.

4. *Spasm of the Sphincter* (sphincter vesicæ) in chills, cystitis or when foreign bodies lodge in the bladder ; also, in reflex manner, in colic and tetanus.

Urinary Calculi.—The development of urinary calculi are of great surgical importance, and may have a three-fold origin. They are either formed by the salts contained in the urine crystallizing around some foreign body in the bladder (coagula, sperma, parasites), or are due to an excess of salt in the urine caused by drinking water containing too much lime, or eating food too rich in lime and magnesia (*e.g.*, bran), or excessively nitrogenous fodder (producing uric acid) ; or finally, to formation of insoluble phosphates of ammoniacal magnesia (triple-phosphate) in cases of retention and decomposition of urine in the bladder (cystitis). Dammann describes an epidemic of silicate stones in oxen, which he ascribes to an excess of potatoes in the food. Prietsch mentions a similar occurrence among swine (40 animals). Ebstein's experiments demonstrate that inflammatory processes in the urinary passages pre-dispose to the formation of calculi by supplying the organic foundation for them. Monti found that urinary calculi occurred more frequently among young oxen than among older ones, and ascribes the condition to parasites which are excreted by way of the kidneys and, becoming incrusted with

lime in the renal pelvis, thus lead to the formation of stones. According to Klimmer, stones were found in ¼ per cent. of all the dogs examined at the Dresden Pathological Institute. In their chemical composition the calculi of herbivorous animals are mainly carbonates (also oxalate and silicate calculi); those of the carnivora contain chiefly salts of uric acid) with oxalate and cystine calculi); and those of the omnivora oxalates and carbonates. In all three kinds of animal, phosphatic calculi (phosphate of ammoniacal magnesia) are not rare, occurring after retention and decomposition of urine in the bladder. By giving a dog from four to six grains of oxamide daily, Ebstein and Nicolaier succeeded in producing artificial calculi in a few weeks. Moussu prevented the formation of urinary gravel (phosphate) in lambs by the use of bi-carbonate of soda (8–60 grs.).

Symptoms.—These show themselves either in an entire suppression of urination (ischuria) or an imperfect and difficult passage of water accompanied by pain (dysuria, strangury). An accumulation of urine in the bladder produces in the more sensitive animals, such as horses and dogs, very early signs of discomfort and pain, whereas, in the indolent ruminants, especially oxen, several days may elapse before any characteristic symptoms are noticed. Horses and dogs show at first the symptoms of colic (so-called urinary colic), with frequent attempts to micturate. In cattle and sheep such signs of colic are often entirely absent, though perhaps not rarely overlooked, or we observe only a peculiar peristaltic movement and twitching on the peritoneum along the course of the urethra, and also the unusual dryness of the orifice and its terminal tuft of hair. Palpation of the bladder from the abdominal walls or rectum shows that it is very full—unless rupture has already occurred—and the animal evinces pain on its being pressed. The presence also of some obstruction may, at the same time, be demonstrated, such as a calculus, new growth, etc. This may be further confirmed by careful examination of the orifice and course of the urethra, or by introduction of a catheter or sound. In addition to the above symptoms, the animals appear spiritless, cease to eat or drink, their pulse is quickened, and sometimes they break into profuse sweat.

If at this stage they are not relieved, the symptoms increase, and result either in rupture of the bladder with rush of urine into the abdominal cavity, followed by peritonitis; or in diphtheritic inflammation of the bladder, which develops into pyelitis, nephritis or hydro-nephrosis and uræmia. In the former case the signs of pain and disquiet cease suddenly. On exploration the bladder is found quite empty, while the presence

in the abdominal cavity of much liquid can later be detected. With great dulness of the sensorium, the pulse and temperature both rise; shiverings and general muscular tremblings appear; both breath and sweat acquire a distinct urinary smell; the abdomen grows ever fuller; urination ceases entirely, and the animals finally perish from diffuse peritonitis combined with uræmia. Horses and dogs die very quickly, in a few hours, after previous ischuria, lasting one or two, or, at most, three days. Cattle, on the contrary, may lie for eight to fourteen days after the bladder has burst, and have even been known to live four or five weeks. In the latter case the sickness ends with the phenomena of intense gangrenous cystitis, complicated with acute nephritis and uræmia. This termination is, however, less frequent.

Differential Diagnosis.—Retention of urine is as often mistaken for true colic as this latter for the former; especially by laymen, which is not surprising, seeing that true colic is frequently accompanied by a kind of urinary retention and that the bladder, on examination, is generally found more or less full. A differentiation is, however, with due care, not difficult. In true colic the symptoms of a bowel-complaint gradually predominate, while in retention of urine those of an affection of the bladder become prominent in the form of strangury, frequent adoption of the attitude of urination, spreading the legs, active movements of the tail, persistent ischuria or strangury and sensitiveness of the bladder to pressure. There are also other diseases which may give rise to error, *e.g.*, rheumatic hæmoglobinæmia of horses, simple cystitis, nephritis, peritonitis and finally labour-pains. In none of these, however, is the discharge of urine permanently disturbed, nor the bladder for long abnormally full.

Therapeutics.—In the mechanical form of urinary retention, the treatment is entirely surgical (urethrotomy, cystotomy, puncture, circumcision). In the so-called dynamic form, caused by paralysis or cramp of the bladder—usually by the former—we must first attempt to empty the bladder by pressure from the abdominal wall or from the rectum, as well as by introduction of a catheter. Reflexly we may also secure this result by irritating the orifice of the urethra, or by gently stroking the urethra along the perineum. If there be spasm of the sphincter, we recommend plentiful rubbing of turpentine,

or the oils of aniseed, caraway, fennel and camomile upon the abdomen and perineum, as well as internal administration of these oils, together with morphia, belladonna and chloral hydrate. Strychnine, physostigmine and electricity may also be tried in severe paralysis of the bladder.

Incontinence of Urine, *i.e.*, the inability to hold water, is a condition exactly opposed to retention of urine. It is a symptom of several complaints, especially of affections of the spinal medulla, and arises from paralysis of the sphincter vesicæ, rarely from cramp of the detrusor, *e.g.*, in cystitis. The condition is also noticed after prolonged retention of urine; in weakness of the sphincter from old age and in cystitis; after cystotomy, when the muscle of the sphincter has been severed; when it has been destroyed by tumour; after distension of the bladder by stones or of its orifice by polypi. The *treatment* must be regulated by the primary disease. In paralysis of the sphincter it consists in administration of strychnine, the application of electricity, cold rinsing of the bladder, and general strengthening of the constitution.

CATARRH OF THE BLADDER.—INFLAMMATION OF THE BLADDER. CYSTITIS.

Etiology.—The causes of cystitis are primarily irritation of the mucous membrane of the bladder by poisonous and infectious matter, which has been excreted from the blood, such as cantharides, turpentine or bacteria. Under this head comes also the inflammation of the bladder occurring in the course of severe infectious disease. Chill may also be a possible cause. Cystitis also arises from complication with inflammation of an adjacent organ, thus from nephritis, pyelitis, urethritis, vaginitis, peritonitis or metritis. Traumatic influences, such as bruising or crushing the mucous membrane by calculi, by sounds, or by infection through an unclean catheter, may all produce cystitis. Finally, retention of urine in the bladder, as by a dog when kept too long in a room, may cause cystitis, at first catarrhal, later necrotic. Compression, stricture or stenosis of the urethra (paralysis of the detrusor), cramp of the sphincter consequent on rapid decomposition of the urine and the action of its septic matter upon the mucous membrane may all induce this disease. Among bacteria, the bacterium coli commune and also proteus vulgaris are especially connected with the etiology of cystitis.

Post-Mortem Conditions.—These vary according to the degree of the condition. As with other mucous membranes, an inflammation may be either catarrhal, ulcerous, croupous,

diphtheritic, necrotic or hæmorrhagic in nature; it may be chronic, hypertrophic, associated with varicose degeneration of the vessels or phlegmonous, causing submucous and pericystitic abscesses, and even perforation of the bladder; or, finally, it may become a so-called " incrusting " inflammation, that is, complicated with deposit of urinary salts in the mucous membrane of the bladder. In the simple catarrhal form we find the mucous membrane swollen, reddened and covered with a layer of mucus, cast-off epithelial cells, with occasional white blood-corpuscles, in the ulcerous state, this layer consists almost entirely of white blood-corpuscles. The chronic form is distinguished either by thickening, by warty proliferation, varicose enlargement of the vessels of the mucous membrane, or—as is frequent in mares and cattle—by incrustation of the same with lime-salts. In the croupous and diphtheritic forms, membranous deposits and necrotic scurf overlay the mucous membrane, causing ulcers.

Symptoms.—In an acute cystitis the urine is passed frequently, in small quantities, and even in drops, causing more or less pain to the animal. Male animals straddle widely, erect the penis and draw up their testicles. Females press hard, open and shut the orifice and move the clitoris. Unrest and colic are also apparent. Palpation of the bladder from the outside (in small animals), or from the rectum is painful. According to the degree of inflammation there is varying fever, while the consumption of food is interrupted or ceases entirely. The urine on examination is found to contain products of the inflamed mucous membrane: desquamated epithelial scales, white blood-corpuscles, mucus, albumen and, where decomposition of urine has set in, crystals of ammonia, phosphate of magnesia and bacteria. With the carnivora the acid reaction changes to a neutral or alkaline. Finally the urine shows—and most distinctly in purulent cystitis—a more or less thick, slimy or flaky sediment, which is at times entirely purulent, mixed in the croupous diphtheritic form with necrotic pieces of tissue; and in the hæmorrhagic form with red blood-corpuscles. *Chronic cystitis* runs its course not unfrequently without any striking external signs, and is consequently often overlooked, especially in cattle. Careful examination of the urine is therefore important for diagnosis, as in all kidney diseases.

Therapeutics.—The internal treatment of cystitis consists in administration of boracic acid, salicylic acid, benzoic acid, chlorate of potash, tannin, naphthalin, resorcin and creolin. Of the last-named, give to a moderate-sized dog daily, three minim doses. In chronic cases, give internally resinous remedies, viz., turpentine, balsam of copaiba and balsam of Peru. Trasbôt also recommends for catarrh of the bladder, bromide of potash and bromated camphor. The chief treatment of cystitis is, however, external, and consists in rinsing out the bladder with antiseptic fluids, *e.g.*, with a ½ per cent. solution of creolin, or 1 to 3 per cent. of carbolic acid, boracic acid, chlorate of potash, as well as a 1 to 5 per thousand solution of corrosive sublimate, or ½ to 2 per cent. of nitrate of silver. For further details see the text-books on surgery.

Hæmorrhage of the Bladder originates from injuries by calculi, contusion by a fall, pressure consequent on fracture of the pelvis, or some inflammation of the mucous membrane of an ulcerous or hæmorrhagic nature (*cystitis hæmorrhagica*). Acute inflammation caused by chemical irritants, and chronic inflammation concurrent with hæmorrhoidal enlargement of the veins are very common sources. Finally, new vascular growths, in process of suppuration or ulceration, especially papillomata, sarcomata and carcinomata. The *symptoms* consist in admixture of blood in the urine (hæmaturia), which stains it and forms a red sediment composed of blood-corpuscles and fibrinous coagulæ. A larger quantity of blood is noticed after previous severe exertion, when, occasionally, almost pure blood may be passed. On the decomposition of this blood, catarrhal inflammation of the kidneys usually supervenes if the hæmorrhage be long continued. General health is also much impaired, being marked by growing anæmia and weakness. The patient sometimes bleeds to death. In the hæmaturia of cattle the urethra occasionally becomes so clogged with coagulated blood as to cause rupture of the bladder (Mathis). The treatment of hæmorrhage of the bladder is mainly external and surgical, and consists in washing the bladder out with iced water, as well as with solutions of liquor ferri, creolin, alum and tannin. With these we may administer internally ergot, fluid extract of hydrastis, tannin, sugar of lead, etc. Decomposition of the blood in the bladder must be prevented by internal use of boracic acid, salicylic acid, creolin, chlorate of potash, etc., or by rinsing with disinfectants.

Hæmaturia, "bloody urine," or the passing of red blood-corpuscles with the urine, must be distinguished from hæmoglobinuria, which is an admixture of the red colouring matter alone (hæmoglobin). The fact that of late years they have been often classed together has made the differentiation and identification of certain diseases of horses and cattle more difficult. Hæmaturia is not a uniform or independent

disease, but merely a symptom of the most varied disorders of certain organs. As its causes we would indicate : 1. *Injuries to the kidneys* from a blow, heavy fall or contusion, in dogs from being run over, over-exertion, or the intrusion of some foreign body. 2. *Inflammation of the kidneys* (hæmorrhagic nephritis) ; acute inflammations especially, often exhibit slight hæmaturia in their initial stage. Here, also, we may include those nephrites which are excited by animal or vegetable irritants (fresh fir-tree sprouts, cantharides, mercury, bryony, or sharp diuretics), by certain fungi (those of mildew, ergot or mould), as well as by some mineral poisons (mercury, phosphorus). First of all in practical importance is that form of hæmaturia caused by acid plant-stuff, a disease in cattle generally known as "bloody urine" (see "Enzoötic Gastro-enteritis"). 3. *Pyelitis*, especially the purulent form ; also the presence of eustrongylus gigas may produce hæmorrhage. 4. *Cystitis*, both hæmorrhagic and chronic, or the kind which causes enlargement of the vessels. Hæmorrhage of the bladder is a common disease among cattle. 5. *Cystic calculi*, injury of the bladder, cystic-tumours (cancer, polypi, tuberculous and mucous infiltrations), ulcers in the bladder. 6. *Acute infectious diseases*, in the course of which so-called hæmorrhagic diathesis develops, especially anthrax, pyæmia and septicæmia, murrain, pox, distemper of dogs and petechial fever. 7. *Inflammation of the urethra*, urethral calculi. 8. *Prostatitis*, concretions of the prostate. 9. *Constitutional ailments*, in the first place hæmophilia, also leucæmia, pernicious anæmia, etc. 10. *Filariæ in the blood*, producing lesions of the renal vessels, with consequent hæmaturia.—The chief *symptoms* of hæmaturia are the red or reddish-brown colour of the urine, or deposits of red sediment, and microscopical proof that red blood-corpuscles are present. In *differential diagnosis*, hæmaturia must not be confounded with flow of blood from the vagina or uterus (menstruation, metrorrhagia), nor with abnormal discolouration of the urine by medicinal pigments (rhubarb, senna, tar, biliary pigments, santonin, antifebrin).

Tumour of the Bladder.—In the case of the domestic animals, tumours in the bladder are rare. They are generally pappiloma and villous carcinoma ; more rarely, myoma, sarcoma, and fibroma (polypi). The symptoms to which they give rise are bleeding from the bladder (hæmaturia), retention of the urine, and indications of cystitis.

APPENDIX TO DISEASES OF THE KIDNEYS.

HÆMOGLOBINÆMIA OF HORSES.

Black Disury, Azoturia, Lumbago, Hæmoglobinuria.

General Remarks.—Hæmoglobinæmia of horses, as its name implies, has no reference to kidney diseases; for the mere fact that, during its course, a secondary nephritis is sometimes developed, can be no justification for so classifying it, as has erroneously been done. It ought to be classified with the muscular diseases. We deal with it here, because the practitioner is accustomed to refer for so-called black disury among the kidney diseases, and also because a very important symptom (hæmoglobinuria) can be best treated in this group; also we do not wish to separate black disury (or ischury, as it is also called) of horses from the so-called "bloody urine" of cattle, which may be in its nature, either a hæmoglobinæmia or a nephritis, and therefore more nearly allied to the kidney diseases.

Historical Notes.—The hæmoglobinæmia of horses has been recognised from ancient times, and is probably one of the oldest equine diseases known. It has been called variously: "black-water," "wind laminitis," "typhus of the spinal cord," "lumbar typhus," "congestion of the spinal-cord and kidneys," "rheumatic lumbar paralysis," "epizoötic paraplegia," "nervous stroke," "renal stroke," "neuropathic affection of the brachial and lumbar nerves," "lumbago," "lumbar spasm," "azoturia." The veterinary journals in the first part of the nineteenth century mention hæmoglobinæmia, although under another name. In districts where the disease was not prevalent, it was frequently confounded with other complaints; such was the case until very recently in Austria and Denmark. Hæmoglobinæmia was first recognised and clinically described by veterinary authorities in Bavaria and the Palatinate, and, later, in Wurtemberg, North Germany, France and Holland. From the first, however, diverse views prevailed concerning its nature, as the number and variety of the names by which it was known shows. Even now its origin and, more especially, its etiology, is not entirely explained. The following are the chief pathological processes with which it has been considered identical:—

1. *Inflammation of the kidneys*; hence it is sometimes spoken

of as acute Bright's disease. (Hofer, Hering, Bruckmüller, Pflug, Zündel).

2. *Inflammation of the spinal cord* (Haubner, Friedberger, Csokor). Dexler has also recently described hæmoglobinæmia as due to an affection of the spinal cord (alteration in the motor cells of the anterior cornu).

3. *Decomposition of blood,* by the action of infective or toxic material (Kolb, Spinola, Vogel, Bollinger, Eber, Dieckerhoff and others). According to Bollinger, hæmoglobinæmia in horses is the result of a species of auto-intoxication, due to the formation, under the influence of cold or exercise, of a toxic agent ("fermentation," "product of fatigue"), which destroys the blood-corpuscles and produces hæmoglobinæmia. Other authors claim to have found definite schizomycetes in the blood (Cadéac), *i.e.,* streptococci in the subarachnoid space and in the kidneys (Liguières), to which they ascribe the disease. (Nocard, Cadiot, Lucet, Jobelot, and others, did not find streptococci. Nowak says that the streptococci invariably present in the cerebro-spinal fluid after death have no etiological connection with hæmoglobinæmia).

4. *The dissolution of the red blood-corpuscles* by the products of increased retrogressive muscular metabolism, especially by urea and its extractives (Siedamgrotzky and Hofmeister).

5. *Over-production of red corpuscles,* resulting from inaction and an over-stimulating diet (McFadyean).

6. *Rheumatic affection* of the muscles of the buttocks and loins, with degeneration, inflammation, œdema, and fatty degeneration (Weinmann and Lechleuthner, the North German veterinary authorities).

7. *Myositis rheumatica,* with solution of the pigment of the muscles and its absorption by the blood (Fröhner).

Etiology and Pathogenesis.—We first notice an inflammation of the muscles of the hind limbs, produced by cold, whereby, amongst other things, a dissolution of the muscular colouring matter (identical with hæmoglobin), and its passage into the blood, take place. We therefore consider Fröhner's name of "rheumatic hæmoglobinæmia," as by far the most suitable for most cases of the disease. Those rarer cases of hæmoglobinæmia, however, which are brought on by over-exertion, must be regarded as parenchymatous myositis, and in contra-distinction to *rheumatic,* described as "*spontaneous*" hæmoglobinæmia.

1. The commonest cause of hæmoglobinæmia in horses is probably a preceding chill. They acquire a pre-disposition to it, by standing some days in warm, steamy and ill-ventilated stables, being well fed the while, for instance, during holidays, or when lame. Heavy, hard-working horses are most affected. The warm and well-protected stables of richer owners are proportionately more troubled by this disease than the badly appointed ones of the poorer classes, and its appearance in a district can generally be easily traced to the varied stable arrangements. When animals that are richly fed and have been long kept in heated stables are brought out into the cold air, they are much more susceptible to chill than others living amid hardier surroundings.

The manner in which cold acts upon the organism and sets free the hæmoglobin in the body, may be thus explained. As is shown by physiology, metabolism in the muscles increases on any irritation of the sensitive, temperature-nerves of the skin. If then intense irritants be set to act upon the skin, this change of substance in the muscular system becomes greater and may grow to disintegration of the organic albumen, that is, of the muscular substance itself. In consequence of this degenerative muscular change, the products of such metabolism pass into the blood and, with them, also the colouring matter of the muscles. This colouring matter is identical with hæmoglobin. Hæmoglobinæmia is therefore of myogenous, and not of hæmotogenous, origin. The startling effects of such a chill are explained by the fact of the animals having been rendered unfit to encounter it by days spent in a warm atmosphere. The reason why the hind-legs are chiefly affected is that they, with the croup and loin muscles, have most to do with a horse's movements, and are the parts least covered up to protect them from cold (Fröhner).

2. After excessive muscular exertion (over-straining with draught-horses; long-distance drives and rides; reconnaissance expeditions; transport; prolonged railway journeys; nervous excitement after long operations, after parturition, or when caught up in the halter-chain), a severe, acute muscular degeneration (*myositis parenchymatosa*), especially of the psoas muscle, the longissimus dorsi, the gluteal muscles, the quadriceps femoris, and the anconeus, is frequently observed. It may be general, or confined to a single group of muscles; may occur on one or both sides; and may, or may not, be accompanied by hæmoglobinuria. Respecting symptoms, course and *post-mortem*

appearances, this "spontaneous" hæmoglobinæmia is the exact clinical counterpart of the rheumatic form (Fröhner, Bergstrand, Haase, Dages, Leipziger, Warncke and others).

3. In a few cases the agency of chill appears to be absent, although, as is known, horses may take cold when standing in their stalls. For lack of a more definitely-known cause, these may be termed "infectious," that is, "toxæmic," hæmoglobinæmia.

Chill is acknowledged by the great majority of observers to be the chief cause of the disorder. It cannot be supposed to be by direct separation of the red blood-corpuscles that it acts, seeing that the degree of cold experienced is far less than that required for the disintegration of blood (freezing). From Lassa and Nassaroff's experiments it has been shown that parenchymatous muscular degeneration, with reduction of colour in the muscles of the skeleton, may be effected by sudden chill. It is known that in horses intense myositis occurs after chill, or during severe winter cold. Moreover, a decided "cold-disease," the so-called "paroxysmal or winter hæmoglobinuria," which occurs in man, is caused entirely by cold and closely resembles black disury. According to Rosenbach, it may be experimentally produced by cold, *e.g.*, by a cold foot-bath, and its symptoms coincide remarkably with those of the above-named ailment. That the amount of free hæmoglobin in the blood is greater in rheumatic hæmaglobinuria than that in the blood of healthy horses, has been proved by Schindelka's experiments with the hæmometer. This remarkable state of things can only be accounted for on the assumption that, in rheumatic hæmoglobinæmia, to the ordinary hæmoglobin already present in the blood, are added a large proportion of another substance of a similar colour. This must be the muscular colouring-matter, which is identical with hæmoglobin.

Post-Mortem Conditions.—The chief changes found in horses which have succumbed to hæmoglobinæmia are in the muscles and blood.

1. *The muscular changes*, which are seen chiefly in the muscles of the croup and thigh, consist, when viewed by the naked eye, in œdematous swelling and a paleness of colour. Microscopically examined, they show a granular opaqueness, a splitting up into layers, hyaline degeneration and loss of transverse striation. All muscles are not equally affected, and it is not restricted to those of the psoas and croup alone. Thus, Siedamgrotzky found the bodily muscles in one case decolourised and degenerated, as follows:—Primarily the loin or psoas muscles, the thin adductor, the posterior part of the other adductors of the thigh (fish-bone muscle), the large and broad thoracic muscles; secondly, the four-headed extensor of the lower leg, and thirdly, the anterior portion of the adductor of

the thigh, its adductor, the gluteus muscle and the longissimus dorsi. Zschokke found that hyaline degeneration of the muscles was never absent in hæmoglobinæmia and demonstrated its presence four hours after the commencement of the attack. He observed that the cut surface of the muscles became brick-red shortly after exposure to the air. The nature of these muscular changes must be regarded as an inflammatory degeneration of the fibres. Similar variations are sometimes perceived in the muscles of the heart. Occasionally small hæmorrhagic foci are found between degenerated muscular fibres, especially in the psoatic muscles and the patella extensors (*polymyositis hæmorrhagica*).

2. The most important changes in the *blood* are loss of normal colour and an appearance resembling varnish or tar, and no, or only slight, coagulation. Blood drawn from the living animal generally separates on coagulation, the serum being reddish (because it contains hæmoglobin); this is lacking in some cases, probably when the hæmoglobin has been already excreted from the body. According to the investigations of Siedamgrotzky and Hofmeister, the blood is very rich in urea and in extractive substance, the products of the increased muscular metabolism. We find under the microscope an immense number of hæmatoid crystals diffused in the blood.

Besides these more important changes, which are observed in every case examined, certain secondary affections of the internal organs must be named. When, for instance, the muscular hæmoglobin has reached the arterial system, the body at once endeavours to extract it again from the blood by the apparatus at its disposal. To these belong primarily the spleen, which receives the dregs of the red blood-corpuscles, and becomes consequently swollen ("spodogenous" tumour of the spleen, according to Ponfick); then the liver, which also is usually more or less swollen, and the red medulla of the bones, which is first found infiltrated with sugar and dyed an intense black-red. This swelling and infiltration of the red medulla, which are (though not always) noticed in the larger bones (femur, humerus, radius, tibia) must not be regarded as osteomyelitis. Excretion of the hæmoglobin through the kidneys, or hæmoglobinuria, begins only when the amount of free hæmoglobin circulating in the blood exceeds one-sixtieth part of the total hæmoglobin of the body. In the latter case a hæmoglobinous congestion is produced in the kidneys, to which further changes, and especially acute parenchymatous nephritis, may

be added. Finally, hæmorrhage occurs occasionally in single organs (spleen, kidneys, etc.), and septicæmia may easily develop in connection with decubital gangrene.

Symptoms.—The signs of rheumatic hæmoglobinæmia in horses are in general as follows:—In most cases the animals, having stood idly and well fed in their stalls for some days, where the air has been very warm and ventilation bad, on returning to work shortly begin to manifest irregularities in their movements usually within from a quarter to half an hour after starting. In mild cases, the symptoms suggest rheumatism. The animals are stiff, or even lame, in the hind limbs especially, or in one hind or fore-leg. At the same time partial sweating breaks out. In severer cases the horses stagger on their hind-legs, have a stiff and insecure gait, there is laboured propulsion of the hind limbs, the angles of the joints remain more widely open, so that the whole limb appears longer than usual; and the hoofs are dragged on the ground. Then the animals knuckle over at the pastern, etc., tremble, sweat, have difficulty in preserving the equilibrium of their hind limbs, and, not unfrequently, fall down, as though struck by apoplexy. When on the ground, they make frantic attempts to rise, show signs of difficulty of breathing and of terror, and are occasionally bathed in sweat. They can then only be transported to their stables on a wagon or sleigh.

On *palpation* of the paralysed hind parts, the muscles are found very hard, tense and painful, especially those of the croup. The skin of this region is higher in temperature and swollen like a board (œdematous infiltration of the perimysium, subcutis and cutis). The sensibility of the parts to touch, to pin-pricks or electricity, is often unimpaired, and is sometimes quite suspended.

Occasionally, symptoms of a circumscribed, localised inflammation of the muscles with myogenic paralysis are observed. A one-sided paralysis of the patella extensors, with collapse of the hind-legs when the animal is carrying a heavy load, is the form most frequently seen. More rarely, the elbow-extensor is affected, and the elbow-joint gives way when an attempt is made to load the animal. Varying from these conditions, we sometimes notice paralysis of other parts of the body, such as the fore-part, or, but less frequently, one fore-leg, the shoulder muscles, upper neck-muscles, etc. According to Bouley, among ten cases, the left hind-leg was first affected. In one case observed

by Lippold, instead of the usual knuckling over, the fetlock was so over-extended that the hoof and shoe were pushed completely forward, the animal standing upon its pastern-joints. Sometimes the tail is held stiffly. We have often seen agonised contractions of the muscles of the abdomen, and the animals have moaned with pain.

Another striking symptom is the hæmoglobinuria, which appears in all severer stages of the malady, and which was formerly considered a pathological and invariable sign of the disease. It is evident, however, from pathogenesis, and has been confirmed by much experience, that hæmoglobin may be absent from the urine, in mild cases, or in those with abortive course, a fact which proves how unsuitable was the former name of hæmoglobinuria for the disease. The urine *which contains hæmoglobin or methæmoglobin*, is characterised by its dark-red, at times ruby-red colour, or varies from a dirty-brown to an almost inky-black colour. Regarding the reaction of the urine, we must affirm, in opposition to other authorities, that in milder cases it almost always remains alkaline, and even in severer cases, sometimes permanently. Thus, in twenty-five carefully tested cases, five of which ended fatally, and which on the average were all severe, we once only found an acid urine (=4 per cent.), which was very rich in phosphates. According to French practitioners, the urine should contain sugar. The specific gravity is in many, even severe cases, found to be quite normal. From these facts we conclude that in hæmoglobinæmia of horses the urine possesses no diagnostic importance.

There is usually no rise in internal temperature, even in very severe cases. Thus, among 25 cases, we found 20 without any such rise; in four the temperature exceeded 102° Fahr., and in only one was above 104° Fahr. The pulse, on the contrary, is usually quickened. Thus in the above 25 cases, we found no acceleration in four cases; in 16 the rate exceeded 60 a minute, and in five was about 50. It sometimes rises to 70 or 80 or more, being then hard and jerky. The visible mucous membranes are usually redder and of a dirty colour (signs of blood-dissolution). The bodily temperature is unevenly distributed, the extremities being cold. In consequence of the considerable change in the blood, decubitus often appears when the animals have lain ill only a short time (haunches, edge of zygoma, etc.).

In the digestive organs the diminution, or even suppression, of peristalsis is in very many cases very marked. No other

changes occur, the appetite being normal, or even increased. Coincident with the suppression of peristalsis, there is retention of urine in the bladder, which can usually be remedied by pressure through the rectum. The difficulty of breathing, which accompanies the course of this disease, has been already mentioned.

The sensorium is unaffected, and disturbance occurs only when there is complication with nephritis. Thus, in one case we noticed uræmic symptoms, which manifested themselves in much dulness of the sensorium, and in epileptiform spasms. The cerebral disorder and signs (resembling staggers, which some practitioners have noted), may be thus explained. Such indications are, however, secondary.

Course and Duration.—The course is generally acute, and even subacute. Recovery in mild, abortive cases, may take place in an hour or two, or usually within two or three days, in which case it is often a complete one. We must not, however, forget that the course of this disease is occasionally marked by periodical increases of violence, and after apparent improvement a relapse may occur. For instance, a horse may fall ill several times in a year, or for several successive years on the same day, if kept a few days in its stall. Occasionally the same animal will be ill several times in a few weeks. A simultaneous affection of several horses in the same stable has also been observed. Evidently, no immunity is gained by having once passed through the disease, but on the contrary, a predisposition to further attacks. This has been noticed in paroxysmal hæmoglobinæmia of man, which has therefore been called "periodical," or "intermittent," hæmoglobinæmia.

In severe cases, recovery often leaves a paralytic condition of the extremities behind. Either a paresis of both sides of the hind quarters remains for weeks, months or even years, so that the hind limbs appear lame, the feet drag and the gait is uncertain; or else a permanent paralysis of the extremities and of certain groups of muscles is developed on one side, with muscular atrophy. Paralysis and degene ative atrophy of the patella extensors is of considerable practical importance, because, in the greater number of cases, it is incurable. Although often wrongly called "crural" paralysis, it is undoubtedly of myogenic origin. Quadriceps paralysis may continue unchanged for many months, it being finally necessary to slaughter the animals as useless. In ex-

ceptional cases, a cure at the end of six months has taken place. According to Thomassen, in single atrophy of the patella muscles, a cure may be expected in by far the larger proportion of cases, even where there has been no sign of improvement for a longer period than six months. Sometimes there is a high degree of atrophy of the gluteal muscles. In rare cases paralysis of the penis may remain as an after-effect. We also saw a case in which chronic meningitis spinalis, with paresis of the hind legs, of the bladder and of the bowels, occurred as a sequel of hæmoglobinæmia. On dissection we found, as a very rare complication, inflammation of the spinal meninges on the cauda equina.

When the sickness ends in death, the paretic conditions grow more and more intense, and pass finally into complete paralysis. The animal gets more restless, the breathing becomes dyspnœic, and decubitus constant. Consciousness grows gradually duller, and death ensues with the appearances either of carbonic acid poisoning, of paralysis of the heart, or of uræmia.

Prognosis.—This differs according to the intensity of the symptoms. It is always well, even in apparently slight cases, to consider the possibility of relapse or periodical recurrences, and to form an opinion with caution. When the animal is unable to rise and cannot even be lifted by the use of slings (complete paralysis), the forecast is unfavourable, although horses occasionally recover after lying thus for days. The mortality varies much according to race, conditions of weather, individual disposition and the degree of sickness. Sometimes the patients recover one after the other surprisingly well and quickly. At other times we may have a whole series of deaths to record. For this reason the mortality estimates vary largely. Bay estimated that 70 per cent. died out of 368 cases observed. Stockfleth gives 50 per cent.; Bouley, 60 per cent.; Grimm, 40 per cent. We reckon the proportion at the Stuttgart Veterinary Institute at 40 per cent., but at the Münich Veterinary Institute it is only 20 per cent. In the Prussian army the percentage over four years was from 40 to 50 per cent. In general we may set it from 20 to 40 per cent., but in cases of pronounced paralysis at from 50 to 70 per cent.

Differential Diagnosis.—A number of complaints have to be taken into consideration, with which hæmoglobinæmia

may be, and often is, confounded. The most important are :—

1. *Colic*, particularly the rheumatic colic caused by chill, which, owing to its painfulness, the accompanying suppression of peristalsis and frequent persistent lying down, closely resembles hæmoglobinæmia. The previous history of the case, particularly of long standing in the stall, is a good guide, when combined with examination of the croup-muscles, the urine, and the knowledge that the animal cannot get up unaided.

2. *Thrombosis of the posterior aorta and its branches* (iliac, pelvic and femoral arteries) occasionally produces an aspect closely resembling rheumatic hæmoglobinæmia and is, at the first glance, not easy to distinguish. Exploration through the rectum will at once reveal the presence or otherwise of this somewhat rare condition. In plugging of the aorta, the severer symptoms vanish quickly and usually give place to a complete restoration. They frequently appear without cause or warning, and may be induced experimentally by exercising the animals.

3. *Apoplexy of the spinal medulla and also spinal paralysis and paresis of the hind leg* of traumatic or inflammatory origin, may likewise give rise to confusion. The traumatic mode of development and gradual growth of a meningitis spiralis, as well as absence of any primary foci for emboli with apoplexy, all furnish hints to the observer. The nature of the urine will be especially significant.

4. *Tetanus.*—Mistakes may possibly be caused by the firm, tense condition of the croup-muscles. On more minute comparison of the other symptoms, however, differentiation should not be difficult. The development of tetanus is essentially slower, and hæmoglobinuria is never present.

5. *Muscular Rheumatism.*—Proof of hæmoglobinuria, the patient's previous history, as well as the apoplectiform appearance of paresis or paralysis, assure the diagnosis in all cases. Widespread acute muscular rheumatism in horses without hæmoglobinuria is rare.

6. *Nephritis, Anthrax, Petechial Fever*, etc., are all easy to distinguish with due care.

7. *Mycotic inflammation* of the bowels, and other toxic conditions which are accompanied by symptoms of paralysis, appear to have been sometimes confused with hæmoglobinæmia (" infectious " or " toxæmic " hæmoglobinæmia).

8. *Fractures, i.e.*, of the dorsal and lumbar vertebræ, rupture

of the muscles, and paralysis of the peripheral nerves, are, upon examination, easily distinguished from hæmoglob næmia.

Therapeutics.—In the treatment of hæmoglobinæmia of horses prophylaxis plays the first part. The disease can, we believe, be prevented by exercising the horses daily, even on holidays, and by feeding them less liberally on such days. They are thereby hardened against any weather, and relapse is especially avoided. This treatment must be aided by rational ventilation and heating of the stable.

When the disease has broken out, it is first of all necessary to get the horse to its feet and, if possible, to put him in slings, as, when lying down, decubitis and hypostasis of the lungs quickly develop with fatal effect. We must, however, draw attention to the fact that horses which are unable to support themselves when slung run great risk of quickly choking. In all cases which, by reason of extensive blood-dissolution, develop great difficulty of breathing or abnormal acceleration of the heart, it is well to bleed the animal freely. Our opinion is confirmed by others, but we must remember that the blood is sometimes very thick and flows badly. If, as often happens, paresis of the bladder produces retention of urine, attempts must be made to empty the bladder, either by pressure of the flat hand from the rectum, or by introduction of a catheter. In order to assist extraction of the hæmoglobin through the kidneys, we must add as much water as possible to the drink, and seek to stimulate the action of the skin by friction, external applications of spirituous and stimulating agents, such as spirits of camphor with oil of turpentine, and that of the bowels by purging and even drastic remedies. Physostigmine (¾ gr. to 1¾ grs.), arecoline (1⅛ gr.), and barium chloride (8 grs.) are all recommended on account of their rapid action. We have found arecoline of the utmost value in a large number of cases and our experience is confirmed by that of the French veterinary surgeons. Hoffmann, Metzger and Kas advise anti-spasmodics (morphia, bromide of potassium); Dieckerhoff recommends large doses of bi-carbonate of soda (3½ to 7 ozs. per dose, 1 lb. 1½ ozs. per diem); Röder and Peter advise preparations of iodine; Hink, large doses of sugar (1 lb. 1½ ozs.); Döttl, chloral hydrate (⅞ oz.), and calomel (⅛ oz.). The after-paralysis is best treated by means of massage, exercise, electricity, the administration of veratrin in doses of 8 grs., and of caffeine in doses of 30–75 grs. Dietetic treatment consists in meal-drinks, bran-mash, carrots,

green food or good hay. Richer food, especially oats, should be avoided. The convalescents must be gradually brought into exercise.

Sub-acute and Chronic Hæmoglobinæmia.—Utz and Vaeth describe a peculiar sub-acute form of hæmoglobinæmia, in which the general symptoms do not appear suddenly, but gradually, so that the animals are not known to be ill until the third or fourth day. Utz also noticed muscular swellings on the masseter, followed by difficulties of mastication (trismus). Friedberger witnessed marked symptoms of tetanus in two cases of hæmoglobinæmia. Höhne describes a case of chronic hæmoglobinæmia.

HÆMOGLOBINÆMIA OF CATTLE AND OTHER RUMINANTS.

(*Hæmoglobinuria, Bloody Urine, Red Water, etc.*).

Definition.—Much light has, by recent experiments, been thrown on the nature of this disease of cattle, which, in older veterinary literature, is described under various local names. From the misguiding general term o bloody urine we must exclude enzoötic gastro-enteritis, with its complication of nephritis hæmorrhagica, as well as hæmaturia—a symptom referring to nephritis. We have treated this latter disease in our chapter on poisoning. Current literature as well as our own observations, have conclusively proved that bloody urine is not to be regarded as hæmaturia, but as hæmoglobinuria. It is for this reason that we include hæmoglobinuria of cattle in the same group with the hæmoglobinæmia of horses.

Etiology.—The hæmoglobinæmia of cattle originates from various causes. Modern investigation shows that it is essential to distinguish between two forms of the disease, namely: infective or epizoötic hæmoglobinæmia, caused by micro-organisms; and rheumatic hæmoglobinæmia, resulting from cold.

1. *Epizoötic hæmoglobinæmia* in cattle is due to the presence of parasites in the blood (piroplasma or pyrosoma), which are a variety of protozoa and are carried by ticks. They were first shown to give rise to hæmoglobinæmia in cattle in connection with the tropical disease known as Texas fever (America, Cape, East Africa, Transvaal). The cause of this exotic form of the disease is the ***piroplasma bigeminum*** (***pyrosoma bigeminum***), a protistic blood-parasite discovered by Smith which is conveyed by the cattle-tick Boophilus bovis s. Rhipicephalus annulatus. (See chapter on "Texas Fever," Vol. II.)

The form of epizoötic hæmoglobinæmia which occurs among cattle in Europe (Roumania, Italy, France, Finland), and in Germany in particular, is, according to the latest investigations, also due to piroplasma (*pyrosoma bigeminum*), and is conveyed by a tick (*ixodes reduvius*, identical with ixodes ricinus, the common dog-tick), in the same way as Texas fever. The piroplasm, which may be pear-shaped, ring-shaped, or attenuated, invades the red-corpuscles and inoculation takes place by the agency of the tick, through the wounds which it causes in the skin. The disease may be conveyed by larvæ hatched from the eggs of ticks which have sucked the blood of infected animals. Hæmoglobinuria may be induced experimentally by the inoculation of healthy animals with blood taken from diseased ones. It has been proved that parasites in the blood of infected animals may remain living and capable of transmitting disease 531 days after the animal has recovered. They may winter in the body of an animal, living from the pasture-season of one year to that of the next, and in this way provide the ticks with infectious matter, affecting an entire herd of cattle; or, whilst developing, may remain the whole winter in the tick. Blood was found to be infectious after being kept for 60 days, and showed hypinosis at a very low temperature. It is certain, however, that piroplasma in the flesh of infected animals perish twelve hours after slaughter. They were, however, found after nine days in blood of cattle which had been slaughtered, and had not been bled in the ordinary manner (Kossel, Schütz, Weber, Miessner).

Historical Notes.—In the older veterinary literature, infectious hæmoglobinuria in cattle is generally described as "bloody urine," and was ascribed to "bloody urine plants," and also to a "miasma, resembling malaria." After the discovery, by Smith and Kilborne in America (1893), of the piroplasmon as the cause of Texas fever, blood-parasites were identified as the source of bovine hæmoglobinuria in Europe, by the following authors: Starcovici, in Roumania (1893); Krogius and von Hellens, in Finland (1894); Weisser and Maassen, in Hamburg, in the case of an ox imported from North America (1894); Sanfelice, in Sardinia (1895); Celli and Santori, during an outbreak of cattle-malaria in the Roman Campagna (1897); Ziemann, in Lombardy (1898); Jackschath, Kröning, Ziemann, Nevermann, in North Germany (1899 and 1901); Lignières, in France (1900); and Kragerud, in Norway (1901). In 1899, ticks (ixodes reduvius) were shown to cause

hæmoglobinuria in cattle in Finland. Finally, in 1900–2 searching investigations into the etiology of hæmoglobinuria in cattle in Germany were undertaken by Kossel, Schütz, Weber, and Miessner, the result of their experiments being published in 1903

2. *Rheumatic hæmoglobinæmia* originates in cattle, as in horses, from chill. It is well known that it usually occurs in cold, wet, stormy seasons, when animals are driven from sheltered quarters too early to pasture, while it does not appear when they are turned out later in the season. Hæmoglobinuria may be brought on, even in the byre, by drinking-water which is too cold (Rychner), or, at pasture, from very cold springs (Zündel). In Galicia, according to Röll, the disease is most frequently found in cold mountainous districts. Dötter goes so far as to call bloody urine a "rheumatic condition." Utz also ascribes the disease to chill and has known it to break out among stalled animals during periods of intense cold. Krug saw hæmoglobinæmia occur in a draught-ox on the same day that it made its appearance among the horses. Härtle describes the exact clinical counterpart of equine hæmoglobinæmia in the case of an ox which, after standing for a week in the stall was put to ploughing on a very cold day. The symptoms were hard swelling of the muscles of the buttocks and thighs, *deranged motor power*, and coffee-brown urine. According to Hink the "pasture-sickness" (Weiderot), which frequently occurs in the Black Forest, is a form of hæmoglobinuria strongly resembling the black disury of horses. It principally attacks the young males, in the first season they are turned out to grass, and is brought on by chill, the ravenous consumption of very cold or wet fodder, and by unusual exertion. Saur saw a case —of a well-nourished milch-cow—in which general rheumatism was complicated with hæmoglobinuria, due undoubtedly to chill. Baer and Kurtz observed all the symptoms of equine hæmoglobinæmia in a heavy draught-ox, which had been in the stall for six days and on the seventh was put into harness in very inclement weather. The hæmoglobinuria, the sudden paralysis of the hind quarters, and the wood-like swelling of the croup muscles, were all very pronounced. In four oxen who were turned out to graze in cold and stormy April weather, Notz observed stiffness of gait, symptoms of paralysis, muscular swellings and hæmoglobinuria. *Post-mortem* revealed degenerative myositis of the buttock, psoas, thigh and shoulder muscles, exactly similar to that found in rheumatic (Myogenic) hæmoglobinuria in horses.

3. *Toxic hæmoglobinæmia*, which is by no means uncommon, is due to poisoning by fodder infected with fungi, by rotten fodder, dregs from distilleries, weeds from vineyards, etc.

Occurrence.—In Europe, hæmoglobinæmia occurs most frequently among cattle, also among sheep and goats, and in by far the larger number of cases appears enzoötically in pasturing cattle, more rarely and sporadically in the stable, by the introduction of infected ticks into the fodder or litter. This disease, which was known to the Greeks and Romans, is spread over all Europe, but appears to be diminishing with the advance of land cultivation. At present it especially attacks herds in Alpine districts and in the Northern hills and plains. In Germany, it is most prevalent in Prussia and Oldenburg. As in the case of pica, the farms where it is rife have a special designation; and are known as "red-water farms." They are generally in low-lying districts, surrounded by woods and over-grown with alder-bushes, conditions which favour the development of the ticks.

Females and young animals are chiefly affected, also milch-cows; males less so. Several observers have noted the important fact that cattle born and reared on the spot show much greater power of resistance than those newly imported, the latter being attacked suddenly and with great severity. Immunity, however, appears to be rather acquired than inherited. Calves resist the disease better than full-grown animals, and they thus acquire a certain degree of immunity which is increased with every year that they are turned out to graze and exposed to new infection. Provided that the power of resistance be not impaired by external conditions, this degree of immunity suffices to prevent the sexually mature piroplasma, present in the blood of an affected animal, from undergoing the process of reproduction. Certain individuals also seem more predisposed. It often happens that the same cow is affected twice a year, viz., in spring and autumn, and even several times during one term of pasturage. It appears to be especially a spring complaint, and afflicts particularly such herds as have passed the winter on short rations and in crowded byres, and been then driven out early in wind and cold to feed on pastures as yet hardly grown. The disease is fixed and stationary on cold, windy, shaded pastures that are also poor, peaty and marshy. It usually breaks out at the end of the first week or the beginning of the second after arrival—or even sooner—and lasts until about the middle of summer. According to Gerlach, it may

occur in from eight to ten days after the animals have been removed from the place. The complaint is not, however, confined to spring, but has been known to appear in the middle of summer, after cold, wet weather. In the Alps it is said only to occur upon slate rock and not upon chalk formations (Lechner).

It is a noteworthy fact that the disease may be carried from infected neighbourhoods into districts free from disease by apparently healthy animals.

According to Kossel, Schütz, Weber, and Miessner, bovine hæmoglobinæmia has been observed not only in Prussia and Oldenburg in Germany, but in Bavaria, Saxony (very rare), Wurtemburg, Baden, Hesse, Mecklenburg, Brunswick, Schwarzburg-Rudolstadt (rare), Schwarzburg-Sonderhausen (rare), Schaumburg-Lippe (rare), Lippe-Detmold (rare), and in Alsace-Lorraine. In all these provinces the same blood-parasite is recognised as the origin of the disease. In districts where the animals are mostly kept indoors, the malady rarely occurs; and then it is usually among the cattle belonging to foresters, which are the only animals allowed to graze in the open. Hæmoglobinæmia causes the heaviest losses in those parts of Northern Germany, where it is customary to turn all the cattle out to grass. Animals of all ages and sexes are attacked, but calves generally make a better recovery than older cattle. In Baden, in the Black Forest, where cows are kept in the byre, while bulls and oxen are turned out to graze, the latter only suffer from the disease. It has been frequently noticed that animals reared in an infected neighbourhood are less subject to disease than those imported from healthy districts. In exceptional cases, it may occur among stall-fed animals, being introduced by forest-litter. The disease is more prevalent in the spring or early summer (May-sickness), according to the time when the animals are turned out to grass. There are severe outbreaks, ometimes, in the summer and autumn, which may occur as late even as November. In character, the disease is strictly local; the cattle on one part of an estate may sicken, while those grazing over a different pasture remain immune. Forest pasturage, grazing ground skirting on a forest, and boggy meadows near the sea-shore are especially dangerous. The first cases develop fourteen days after the animals are put on the infected pasturage.

Development of Cattle-tick.—Experiments undertaken by Kossel, Schütz, Weber and Miessner go to show that the

cattle-tick (ixodes reduvius, identical with ixodes ricinus) is found in all parts of Germany where hæmoglobinuria occurs. Unlike the tick which occasions Texas fever (Rhipicephalus annulatus), and which may go through all the stages of its development upon one and the same animal, ixodes reduvius twice leaves its host for the purpose of sloughing. This occurs after 3–5 days, when it is fully gorged. Ticks may attach themselves to cattle both in the larva and nymph stages, as well as when they have attained sexual maturity. All three forms may be found at the same time on one animal. The larvæ and nymphs are usually seen on the head (round the mouth, eyelids, ears) and on the udder, while the full-grown tick is noticed on the flanks, the inside of the thighs, the neck, the udder and the perineum. The larvæ, nymphs and females only suck blood, not the males. In some parts of Germany cattle-ticks occur in such enormous numbers that they are collected by the bushel in the evening by the children of the place. Where the tick bores into the skin, circumscribed inflammation, accompanied by irritation, is set up. Ticks in all stages of development occur in infected pastures. They are found chiefly in the grass and bushes on damp, boggy places, and their larvæ sometimes collect in large numbers on the tips of blades of grass, where they fix themselves, and wave their front pair of legs about in the air. They may hibernate in any one of the three development stages. Although they occur plentifully in the woods in spring, summer and autumn, they hibernate on the approach of winter, emerge in April, and, being ravenous, attack the grazing cattle and gorge themselves with blood. Hibernation does not impair their infectious power.

It usually requires five months for the development of a generation. The sexually mature tick, about a week after it has dropped gorged from its host, lays 100–1,000 eggs. From these, at the end of a period averaging six weeks, very small larvæ, provided with three pairs of legs only, emerge and seek the tips of grass-blades. At the end of another three weeks the larvæ change into nymphs, furnished with four pairs of legs, and after a further eight weeks become mature ticks. The male tick is dark-red in colour, furnished with a long back-shield; the female, yellowish-red, half only of the back being covered by the shield.

Hæmoglobinuria has been experimentally induced in cattle in various forms and degrees, by means of larvæ hatched from the eggs of sexually mature ticks which had dropped off gorged

from infected animals. From the time the larvæ were applied, until the appearance of the first parasites in the blood, an interval of eight days elapsed. It is probable that the disease-germ (*pirosoma bigeminum*) completes its development in the glands of the larva, a certain time after the latter emerges from the egg. In this, it differs from the malaria parasite, which attains the sporozoic form in the egg. In the ordinary course of pasture infection, the disease appears to be chiefly conveyed by the larvæ, but recent experiments have shown that infection may also be carried by cattle-ticks while in the nymph-stage.

Symptoms.—The symptoms of hæmoglobinæmia are: fever; the passage of urine more or less red in colour, but not containing blood-corpuscles; stiffness of the hind quarters; and increased sensitiveness to pressure in the neighbourhood of the loins. The disease usually begins with high fever (104°–105.8° Fahr.), about ten days after inoculation, to which diarrhœa is added, that may even be bloody. Appetite, which at first is suppressed, is later normal, or very slightly diminished, and a general lassitude only, with slight rise of temperature, is noticed. Sometimes within six or twelve hours the urine, at first pale, shows a more or less dark to brown and black-red colour, or even looks like tar. It also contains much albumen or hæmoglobin, in consequence of which it foams on being passed, and, when boiled, stiffens to a dark brown, pulpy or gelatinous consistence. It readily forms a muddy sediment, and in sheep, dyes the wool red near the region of discharge. During the first three to five days the reaction, according to Reuter, is acid, but later becomes alkaline (note the ammoniacal smell of the urine). It is an interesting fact that the disease may occur without any red colour of the urine (Stockfleth), whereby its similarity to hæmoglobinæmia of horses is increased. Urine as just described is passed at first in larger quantity, which then often greatly diminishes, with much evidence of painful micturition (strangury). Upon the diarrhœa there mostly follows on the third day an obstinate constipation during which tympany is sometimes observed. The internal bodily temperature rises to 106° Fahr. or even higher.

To these symptoms motor disturbances in the hind legs are soon added, viz., stiff, constrained and clumsy gait, awkwardness and even pain in lying down or rising, arching of the back, together with an intense sensibility of loins and croup; also slight twitchings or even feeble spasms in the hind parts,

while spiral movements on the perineum along the urethra have been noticed during micturition. Finally, the weakness of the hind parts grows more and more marked, until the animal can no longer stand. In the last stage of the disease œdematous swellings sometimes appear on the head, neck, point of the chest, etc. The mucous membranes are either reddened, or else, during later stages, dyed a dirty or citron yellow. The milk itself occasionally assumes a yellowish-red colour and the icterus becomes general (Gerlach). Meanwhile the pulse grows more rapid but feebler, exhaustion prevails, the bodily temperature sinks, and death ensues with a general collapse of strength. The urine may for some time prior to death have again become clear.

Course.—This is generally acute, but may occasionally be chronic. It is usually acute, but not really severe, if the animal is removed early from pasture, or if the operating causes are only slight. In such cases, the hæmoglobinuria ceases on about the fifth day of illness, the temperature falls, the general debility lessens and the appetite returns. The condition lasts, on an average, 10–14 days, and the termination is usually favourable. Stockfleth reports 23 deaths among 79 observed; Krabbe only 38 among 330—that is, 8 per cent.; and Rudovsky 6 out of 50—that is, 12 per cent. Where the disease takes an unfavourable course, the animals soon die from weakness and inanition, or from chronic emaciation.

Blood.—The condition of the blood taken from the veins of an infected animal is very remarkable. Where parasites are present in large numbers, it becomes watery in consistency, does not coagulate, and appears as a smeary brown-red mass. Towards the end of the disease, when few or no parasites are present in the blood, numerous red blood-corpuscles are to be seen with basophile granulations, *i.e.*, red spots (Kossel, Schütz, Weber and Miessner).

Post-Mortem Conditions.—The appearances are those of general infection and are usually not very characteristic. The carcases are mostly anæmic, the blood very fluid, from cherry-red to inky-black in colour. The blood-serum separated by coagulation is at times red (contains hæmoglobin). General icterus is often present. There are no organic changes, except perhaps an acute intestinal catarrh, a catarrhal inflammation of the mucous membranes of the gall-ducts, and, more rarely,

hæmorrhagic enteritis. The spleen is occasionally enlarged; the marrow of the bones is hyper-plastic; liver, kidneys and heart show cloudy swelling. According to Spinola the muscles are slack and pale. Hink found them light-coloured and slack. Reuter noticed them of faded colour, as though boiled; and in several places, particularly about the neck, shoulders and thighs, saw more or less diffuse, bloody-serous or gelatinous infiltration. Stockfleth ascribes the cause of death to extreme anæmia, which is most frequent, or combined with secondary pneumonia.

Microscopic examination of piroplasma. The examination is made by a magnifying power of 800–1,000 diameters. A capillary in the ear of a living ox is pricked and the small drop of blood which oozes out should be caught on a thin cover-glass, which must be quite free from grease. It should be exposed to the air until dry and then fixed by immersion for twenty-five minutes in absolute alcohol. The film is stained either with Löffler's methylene blue or by the Romanowsky method. There is sporadic violet-red colouring of the chromatin of the parasites, which are normally of a blue colour. They are seen lying as ring or pear-shaped bodies upon the red corpuscles (cp. chapter on "Texas Fever," Vol. II.).

According to Kossel, Schütz, Weber and Miessner, the parasites which give rise to hæmoglobinæmia in Germany are identical with those to which cause the condition in Finland. The micro-organisms which bring on Texas fever, however, appear to be larger, and those found in East Africa show variation of form. It appears from this that there are several varieties of the hæmoglobinæmia parasite.

Differential Diagnosis.—Among diseases which may be confounded with hæmoglobinæmia of cattle are, first, various affections of the kidneys which are accompanied by hæmaturia, especially enzoötic inflammation of the bowels. They are not hard to distinguish, if the urine be duly examined. Anthrax, which also is accompanied by hæmaturia, and perhaps with hæmoglobinuria as well, may cause confusion. In this instance the whole process of the disease, combined with examination of the blood, must furnish the indications needed. Finally, the dark, brownish-yellow urine in liver complaints or icterus may be thought to contain hæmoglobin, a doubt we dispel by proving the presence of biliary pigment.

Therapeutics.—In the treatment of hæmoglobinæmia, prophylaxis is of the first importance, and is best carried out

by avoiding pasture feeding and keeping the animals in the byres; by going over them each day and destroying the germ-bearing ticks; by the employment of anti-parasitics, in the form of washes and embrocations (petroleum, oil, creolin, lysol, carbolic acid); and by improving the condition of the soil by drainage and better cultivation. It is also of great importance that a native-born, greater resisting breed of cattle should be raised. Inoculation with fresh blood taken from infected cattle is said, as in the case of Texas fever, to induce a slight form of the disease and to procure subsequent immunity (Kossel, Schütz, Weber, Miessner).

When once the disease has broken out, pasturing must be immediately abandoned and the sick herd taken back to the byre. The disease itself is hard to combat. First, according to the symptoms, we must treat the constipation with laxatives, and later must attack the diarrhœa by styptic remedies (sulphate of iron, 4–6 drams; alum, 4–6 drams; tannin, 4–6 drams; sugar of lead, 2–4 scr.; opium, 2½–5 drams). Jackschath considers sugar of lead to be a specific when given in doses of 1 drachm; he recommends smaller doses as a prophylactic. Iron may be given with advantage (ferrum pulveratum, 30–75 grs.; ferrum sulfuricum, 75 grs.–⅓ oz.). Evers recommends Hæmoglobin-Merck, given subcutaneously in doses of ½–⅔ oz. in 9–17½ ozs. physiological sodium chloride solution. He insists, however, that at the same time the ticks must be removed and the skin washed with a strong solution of salt and water, and it is undoubtedly to this latter measure that the success of the treatment is due · 38 cattle treated in this manner recovered. In Baden, santonin is employed, either subcutaneously or internally. In Finland, quinine is given in 1 or at the most, 2 doses of ⅔ oz. Kragerud and others declare quinine to be useless in this condition. In view of the prolonged course of the disease and the pronounced anæmia to which it gives rise, blood-letting is to be avoided (Stockfleth, Gerlach). In severer cases a transfusion of blood may be attempted (Stockfleth).

Hæmoglobinæmia in other Animals.—Besides horses and cattle and smaller ruminants, especially goats, this disease has been noticed in other animals, such as mules, dogs (in consequence of chill after poisoning or during distemper), in zebras and also in pigs. Utz found dark-coloured urine, rich in albumen, and also among other symptoms, a yellow colour in the fat, in a pig which had been transported about five miles during a cold of 13–16.6° below zero, Fahr. Chill appears, therefore, to be a factor in producing hæmoglobinuria in the other domestic animals.

CHAPTER VIII.

DISEASES OF THE SEXUAL ORGANS.

PUERPERAL FEVER.

(*Milk-fever, Calf-fever, Septicæmia, Post-partum paresis.*)

Ancient and Modern Views on the Nature of Puerperal Fever.—The acute diseases of the female animal after the birth of her young have always engaged the attention of veterinary surgeons, especially as affecting cattle, as we may see by the numerous names under which such diseases are known (calf-fever, milk-fever, puerperal fever, paresis of parturition, septicæmia, eclampsia puerperalis, and many others). The symptoms noted, however, have been so varied that Hering already remarks: "Almost every description seems to refer to a different disease." No wonder, then, that opinions have differed widely as to the origin and connection of these symptoms; the majority of authors have spoken of various forms of puerperal fever—the inflammatory-septicæmic and the nervous-paralytic—while others recognize one form only, viz., that which is accompanied by paralytic symptoms, and have strictly separated all others, especially that called "inflammatory," and classed them among diseases of the uterus. In later years, following the lead of Franck, authors have almost ceased to speak of two *forms* of calf-fever, but have divided this into two completely separate diseases, according to their nature and their anatomical and clinical aspect.

1. *Puerperal-fever*, or *Septicæmia puerperalis*, the inflammatory calf-fever of older authors, has also been termed phlegmonous or diphtheritic inflammation of the uterus, or gangrene of the uterus, and consists in an intense inflammation of the vagina and uterus with consequent general sepsis, caused by reception of septic matter.

2. *Post-partum paresis*, the so-called paralytic or nervous form of calf-fever, is probably due to auto-intoxication (self-

poisoning), arising from toxic material formed in the uterus during the puerperal state (puerperal intoxication).

I.—SEPTICÆMIA PUERPERALIS.

(Septic or Pyæmic Puerperal Fever.)

Etiology and Pathogenesis.—Septicæmia puerperalis must be regarded as a species of septic poisoning, caused by the agency of certain bacteria and, more especially, by the *streptococci*, the *staphylococci* and the *bacillus coli*. The infection to which these bacteria give rise may be either simple or mixed. The channels of infection are injuries which occur in the course of parturition to the pudenda, or to the vaginal or uterine mucous membranes and, more particularly, to those of the cervix and the carunculæ. Infection is conveyed either from without, by the hands of the veterinary surgeon, by instruments, cords, stable-utensils, litter, ichorous discharges, or the vicinity of sick animals; or it arises from internal causes, such as the decomposition of the after-birth, or the presence of the dead and decomposing embryo. It does not always result from serious injuries caused by a difficult confinement, but, on the contrary, occurs more often as a result of slight epithelial abrasions after an easy and normal delivery.

Puerperal septicæmia occurs among all the domestic animals, most frequently in cattle and dogs, but it is by no means rare among horses. In cattle, it is said to arise 2–3 days after parturition and in forensic practice this traditional notion has its convenient side. Numerous disinterested experiments have, however, established the fact, that puerperal fever may appear at a later date where unfavourable conditions (transport, chill) prevail (De Bruin).

Post-Mortem Conditions.—Except in the rare cases which run a very acute course, and in which no inflammation of the uterus takes place, we find ulcers—so-called puerperal ulcers—on the mucous membranes of the vagina, cervix uteris and uterus upon the sites of the original wounds. These are covered with a discoloured, necrotic or diphtheritic deposit, which has sometimes become detached and been replaced by granulations. Around these ulcers the mucous membranes are œdematously swollen. Here and there the swelling attacks the external genital parts, and extends even to the inner sides of the thighs and belly. The remaining mucous membrane of

vagina and uterus are also in a state of phlegmonous inflammation, which, in dogs, frequently develops into diphtheritic scurfiness (kolpitis and endometritis phlegmonosa crouposa and diphtheritica). In the latter case, it is of a dirty brown, or greenish-black colour, permeated by ulcers and covered with ichorous, fœtid matter. In some spots it is changed into a greasy, necrotic mass, on removal of which the muscles of the uterus are laid bare.

The parenchyma of the uterus is itself œdematous and ulcerated. The lymphatic vessels are charged with pus, the veins with thrombi. Under the microscope, the lymphatic spaces of the muscular layers are seen to be thickly filled with colonies of micrococci. The uterus, moreover, is badly contracted, its walls are friable, flabby, and their muscular fibres in a condition of opaque swelling or fatty degeneration. Sometimes there are abscesses among the muscles (metritis phlegmonosa). The connective tissue around the uterus is likewise œdematous, swollen and infiltrated with pus, and traversed by abscesses (parametritis phlegmonosa). Further, the peritoneum surrounding the uterus, and covering the pelvis, and finally the whole peritoneum are in a state of serous, purulent, ichorous inflammation (perimetritis, pelveo-peritonitis, peritonitis diffusa).

If the condition be sufficiently advanced, we obtain now the aspect of septicæmia and pyæmia, and find purulent pleuritis, pericarditis, ulcerative endocarditis, meningitis, arthritis and hæmorrhagic gastro-enteritis. Furthermore we perceive metastatic inflammation of lungs and kidneys, abscesses in the mediastinum, in the subcutaneous and inter-muscular tissue, and in the soft parts enclosed by the claws; swelling and degeneration of spleen, liver, myocardium, mesenteric and lymphatic glands especially; formation of thrombi in the vessels of lungs, liver and kidneys; ecchymosis in all organs, particularly under the endocardium; greasy, badly coagulated, tar-like blood, which infiltrates the tissues; decomposed red blood-corpuscles. and a rapid decomposition of the body, with much generation of gas.

De Bruin (Geburtshilfe beim Rinde, 1902) classifies the different manifestations of puerperal infection as follows:—

1. *Septic infection:* (*a*) puerperal phlegmon; (*b*) septic metritis; (*c*) acute puerperal septicæmia.

2. *Pyæmic infection:* (*a*) pyæmic polyarthritis; (*b*) metastatic tenosynovitis; (*c*) chronic parametritis.

In addition to these, he groups under one heading the puerperal toxic conditions (poisoning by retained lochia and putrid puerperal poisoning).

Symptoms in Cattle.—Except in the rare cases of rapidly developed general sepsis with very acute course, the disease usually begins with signs of inflammation in the genital passages. First, the mucous membrane of the vagina and vulva is found to be reddened, swollen, higher in temperature and covered with diphtheritic ulcers and ichorous secretion. Occasionally the phlegmonous swelling of the vulva extends to the inner sides of the thighs. On palpation from the right abdominal wall, or from the rectum, the uterus is found to be swollen and painful. Ulcers are frequently found grouped especially about the urethral orifice. Corresponding to these inflammatory affections of the vagina, uterus and urethra are the signs of pain manifested by the animal. It grows restless, wriggles its tail, arches its back, looks backwards, shuffles about, often lies down, contracts its abdominal muscles, presses and strains upon the uterus as though in extreme labour pains, as a result of which a foul-smelling, discoloured, ichorous-looking fluid is discharged, finally it often attempts to micturate, but can pass only a few drops with much pain and effort.

At the same time a great rise of temperature is observed, accompanied by shiverings. The rectum will show as much as 104° to 107.6° Fahr.; and the pulse, 80, 100 and 120 beats per minute, but the latter becomes small, thread-like, and often imperceptible. The extremities, such as feet, horns, ears, and even the dry muzzle feel alternately cool and then very hot. If the animal lie long, decubitus quickly sets in. With these signs, there is complete cessation of both eating and drinking, and also of milk-secretion, accompanied by constipation and slight tympany. The sensorium is excessively dulled, all movement becomes sluggish and uncertain, feebleness rapidly increases, so that the animal cannot stand. At this stage the disease much resembles the aspect of paresis of parturition, but may be distinguished therefrom by the very high temperature, the inflammatory local changes and the lack of the characteristic muscular paralysis in the eye, the pharynx and the larynx.

The course of the disease may vary in three directions. Either death occurs with increase of the above-named symptoms, being sometimes immediately preceded by diarrhœa,

the end in such cases coming in from three, four, or at most six days (more rarely it is apoplectiform, and follows in one or two days); or complete recovery may be made in from eight to fourteen days, with abatement of the inflammatory signs, fever, etc.; or, finally, a chronic condition of illness may develop, characterised by a protracted, wearisome sickness, with great emaciation, hectic fever attacks, extreme cachexia and final exhaustion. Chronic endometritis complicated with persistent discharges (fluor albus), when the mouth of the womb becomes closed, resulting in complete sterility; pyæmic metastasis of the lungs, joints and other organs sets in, as also chronic inflammation of the kidneys, with its consequent phenomena. The metastatic inflammations just named convey the impression of a recurrence of the disease, and may occur several times in succession. If this condition lasts long, hæmorrhagic diathesis may develop, with hæmorrhage in the kidneys (hæmaturia), in the lungs (bleeding at the nose), and in the bowels. We have seen distinct petechiæ in great numbers on the mucous membrane of the nasal cavity, as in the petechial fever of horses.

The mortality in septicæmia puerperalis amounts to from 50 to 70 per cent.

Symptoms of Puerperal Fever in Swine.—These have more resemblance to the paralytic form of puerperal fever in cattle than to septicæmia puerperalis. The disease is marked by a much slighter and less dangerous course, and is commonest among half-English races and those in well-fed condition. Like the paresis of parturition in cattle, it follows chiefly after easy births and after the fœtal membranes have been passed, usually three days after birth. According to Herz, the following are the several symptoms:—The animals show diminished appetite and milk secretion, grunt slightly while lying, have an unsteady gait, lie much, stagger and reel, and, at last, avoid every movement of limbs or head, giving the impression of paralysis; take no notice of their surroundings, wear a dull, mournful aspect, keep their eyelids half-closed and sometimes make rotary movements with the bulbus. The bodily temperature is unevenly distributed, that of the interior being 104–106.7° Fahr., the pulse rises to 140 a minute, the bristles are erected, the skin is hard and dry and somewhat reddened behind the auricles and on the thighs. At intervals a quivering of some groups of muscles is noticed. The vulva and mucous membrane of the vagina are dark red, but only slightly swollen. From the vagina a small quantity of viscid, whitish mucus is exuded. The breathing varies from 20 to 40 per minute and is accompanied by slight groaning. Peristaltic movement is suppressed, and constipation present. The course is acute; recovery usually follows in from one or two days, more rarely in five days. Occasionally weakness of the loins and interstitial mastitis are left behind.

Symptoms of Puerperal Fever in Sheep and Goats.—The animals are dull, refuse food, hang their heads, develop an expanded and sensitive belly, arch their backs, and show persistent straining and colic symptoms, as in labour pains. The visible mucous membranes are reddened, the temperature rises to 105.0° Fahr.; and later a gradual and extreme weakness sets in, with loss of consciousness. The external genitals are more or less swollen, and upon the mucous membranes dark-red streaks and ulcers are seen. The duration of this disease, which is evidently septic, varies from a few hours to, at most, one day, and is generally fatal. It is possible that some of the diseases designated as "gangrene of the uterus" in sheep belong to puerperal fever.

Symptoms of Puerperal Fever in Dogs.—These are those of a septic metritis; swelling of the genitals, bad-smelling and discoloured discharge from the vagina, very high fever, 105.8° Fahr. and more, refusal of food, vomiting, persistent lying, sleepy and comatose condition. The end is mostly fatal within from 12 to 24 hours, with great fall of bodily temperature, or else after 12 hours improvement begins. Chronic endometritis is often left behind, as with cattle.

Symptoms of Puerperal Fever in Horses.—With the horse also the phenomena of this complaint belong to the realm of septicæmia and consist mainly in higher temperature, quick but weak and irregular pulse, unevenly distributed warmth of body, shiverings, loss of appetite, suppressed peristalsis, colic, great stupor, as well as local changes in the genitals. The course is very acute. Koninski states that he saw a case of paresis of parturition in a horse.

Therapeutics.—The treatment of septicæmia puerperalis consists mainly in disinfection of uterus and vagina. The old method of irrigating with carbolic acid (1 to 5 per cent.) may with advantage be replaced by a 1 to 2 per cent. solution of creolin, which possesses the advantage of being non-poisonous, and is more effective, as it immediately removes the bad odour. It is therefore highly commended by many practitioners. Lysol is also good for rinsing out the uterus. Infusions of sublimate (one per 1,000) are suitable only for the carnivora; for the herbivora, and especially for cattle, it is too poisonous, and causes great straining—although other authorities have maintained the contrary. Ergot, or fluid extract of hydrastis, may at the same time be administered internally to produce better contraction of the uterus, and thereby lessen the risk of its mucous membrane absorbing septic matter. Gentle purgatives are advisable to assist the excretion of infectious matter through the intestinal canal. The activity of the skin may also be stimulated by friction. Finally, the high temperature and signs of weakness may be symptomatically treated with pyretics and stimulants. As a prophylactic measure, the strictest cleanliness

(asepsis) must be required from the obstetrician (hands and instruments). It is also advisable to isolate highly pregnant animals from others in less advanced condition, and from such as suffer from abortion, chronic discharge from the vagina, or ulcerous skin affections ; avoid also any chance transmission of the disease by attendants, implements, food, drink or sponges, and disinfect beforehand the byre with creolin, sublimate, carbolic acid, chloride of lime and other means. Finally, secure good ventilation.

II.—POST-PARTUM PARESIS (THE TOXIC OR PARALYTIC FORM OF PUERPERAL FEVER). MILK FEVER.

Etiology and Pathogenesis.—Nothing definite is at present known as to the causes of post-partum paresis. Numerous theories and hypotheses have, however, been propounded, the most important of which may be briefly described as follows :—

1. According to the most generally accepted view, the paresis of parturition is the result of auto-intoxication. The theory originated with Schmidt-Mülheim, who based his views upon the similarity between the clinical aspects of post-partum paresis and ptomaïne poisoning, and assumed the former condition to arise from an auto-intoxication caused by the decomposition of lochia in the uterus. In the first edition of this book, we adopted the auto-intoxication theory and for a long time stood alone in our defence of it. Now, however, the view is held by the great majority of veterinary surgeons in Germany (Albrecht, Eber, Eggeling, Ostertag, etc.). Unfortunately, there is considerable diversity of opinion as to the actual part of the body in which the toxin is formed. Some hold that the uterus is the source of infection ; others, the udder (Schmidt, Kolding, Walther, Thomassen) ; others, the bowels (Kaiser, Knol) ; others again, that the toxin forms in the blood itself and thus passes in every part of the body (Ehrhardt).

2. In France and Belgium (Nocard, Vander Velde,) and to a certain extent, in Switzerland (Zschokke, Guillebeau, Hess) post-partum paresis is regarded as an infective disease, resulting from the introduction into the uterus of micro-organisms. Such are the staphylococci, streptococci, bacillus coli, bacillus œdematis maligni. They lead to the formation of toxins of which the absorption is said to give rise to paresis.

3. To Franck is due the credit of defining the clinical difference between puerperal septicæmia and post-partum paresis. He classifies the latter as a distinct pathological condition, but is wrong in calling it " eclampsia " and in attributing it to circulatory disturbances in the brain (cerebral hyperæmia, cerebral œdema). According to him, the latter arise after parturition as a consequence of the loss of blood from the enlarged uterine vessels.

Aronsohn, Teetz, Zehl, Meier and others have recently advanced the theory that post-partum paresis is due to anæmia of the brain, caused by the rush of blood to the udder. They ascribe the beneficial results obtained by pumping air into the udder and by water infusions, to their mechanical action in emptying the vessels of the udder by means of pressure. This view is untenable for the reason that hyperæmia of the udder always occurs in parturition and hence, according to their theory, anæmia of the brain and consequent paresis would break out in every calving cow.

Harms believes that the condition arises from the entrance of air into the ruptured uterine vessels (æræmia), this leading to partial anæmia of the brain, due to air-embolism.

Occurrence.—Milk fever or post-partum paresis occurs most frequently in cows and is also met with in sheep and goats. Cows which are good milkers are most liable to it, as well as fat, full-blooded and well-nourished animals that have been very highly fed before calving. This is proved by the fact that paresis is rare in districts where the milk is poor; while on dairy-farms, especially among Dutch or fen cattle, it is a source of greater loss than any other disease. Some observers profess to have noticed an inherited predisposition in certain kinds of cattle, and in support of this theory it is stated that, in districts where the disease was previously unknown, it appeared simultaneously with the introduction of a different breed of cow. It occurs by no means infrequently among pasturing cows. It is a remarkable fact that paresis usually follows a quick and easy delivery and for this reason cows rarely develop it after their first calf, the first delivery being usually difficult, but generally after the birth of the third to the fifth calf. Older cows, again, are less frequently affected. The greater number of cases occur either in hot summer time or when the weather changes suddenly. Whether or no chill plays a part in the causation of post-partum paresis is doubtful, but in any case its influence can be only indirect. The condition usually makes its

appearance 12–48 hours after parturition or, at the very latest, at the end of the third day. It rarely occurs during delivery. In a few cases it has been recognized before the birth but never before the appearance of the milk (Müller, Söhngen, Brüller, Winter, de Bruin, Albrecht, Meltzner, Andersen, Monsarrat, Fenimore, etc.). It has been frequently noticed that, after recovery, a predisposition to further attacks remained (de Bruin. Bru, Gassner, etc.). It is also said that keeping the animals too long without water predisposes to post-partum paresis (Zehl).

Post-Mortem Conditions.—These offer little or nothing that is characteristic. The uterus is usually found strongly contracted and without any injury or inflammatory change. The only thing noticeable is an unequal distribution of blood in the body, in that the viscera of the belly appear fully charged, as compared with other organs. In many cases the brain shows nothing abnormal; sometimes the meninges are very hyperæmic and traversed by hæmorrhages. Hyperæmia of the medulla oblongata has been demonstrated, as well as internal and external hydrocephalus with transuded matter resembling broth (sarcohydrocele). The brain is often found to be very anæmic, flabby and collapsed, and its cortex stained a dirty yellow; changes which in other cases are absent. The cavities of mouth, nose and pharynx are sometimes found stuffed with food (paralysis of the swallowing apparatus). The drying-up of the third stomach, formerly regarded as characteristic, occurs in most disorders with prolonged suppression of food-consumption, and is therefore quite secondary. All the other organs appear to be normal.

Symptoms.—The ailment usually begins with signs of slight restlessness; the animals shuffle about, strain to evacuate and strike their feet up against their body. In a few cases pronounced signs of brain irritation primarily appear; the animal becomes excited, bellows and rears. In very rare instances we perceive epileptiform spasms, twitchings in the muscles of the face, grinding of the teeth and spasmodic twisting of the neck. Some observers have remarked a considerable, hard swelling of the udder at the commencement of the malady.

After these initial stages, which are often overlooked and may, indeed, be absent, the essentially characteristic signs of depression and paralysis appear. The animal primarily betrays weakness in the use of its extremities, especially the

posterior ones, staggers when walking, appears dull and powerless, sometimes falls and struggles to rise again, generally in vain. This paresis rapidly extends from the hind quarters to the whole body, muscular weakness obtaining complete mastery. The animal can no longer stand, but lies helplessly on the ground, its legs half doubled up, or, in extreme feebleness, with all four outstretched, in which condition a medical man usually first sees it. It lies in complete apathy and somnolence, its head held sideways, and the neck so bent that it rests on the side of its breast. If we attempt to lift the head, it falls back powerless into its old position. The upper eyelid droops over the eyeball (ptosis), the eyes are shut, and the animal appears to be asleep. On raising the eyelid, the cornea is found to be strangely dull and glazed, as in a dying animal, dry also and insensible, even crinkled and fissured. The pupil is amaurotic and enlarged, secreted tears are collected under the eyelid, the eye-ball is considerably sunken. The tongue hangs loosely from the mouth, owing to paralysis of its muscles and saliva flows out in long strings, unless its secretion be suppressed, because it can no longer be swallowed. When swallowing fluids, we hear a strange, clucking, gurgling sound all along the throat. Breathing is performed with snoring, groaning, rattling and even whistling noises, accompanied by much distension of the nostrils.

On examining the digestive organs we find obstinate and continuous constipation, caused by paresis of the involuntary muscles of the bowels. This intestinal paralysis generally resists the most powerful purgatives, even physostigmine. It is soon followed by tympany and suppression of all intestinal sounds, accompanied by cessation of urination, caused by paresis of the muscles of the bladder. The urine contains albumen and also sugar, according to Nocard, even as much as 40 grammes per litre ($1\frac{1}{4}$ oz. in $1\frac{3}{4}$ pints) (Albrecht, on the other hand, found small quantities of sugar in exceptional cases only). Milk-secretion is likewise suspended, and the udder is relaxed and limp.

In the circulation, we are first struck by the unequal distribution of bodily temperature, the feet, horns and ears especially feeling icy-cold. The pulse is feeble and often imperceptible; the heartbeats are always quickened, numbering from 50 to 70 and later 120 and more per minute. The internal temperature is normal or sub-normal, and raised only in rare cases at the beginning of the attack. High degrees of fever do

not occur in post-partum paresis. If, in any case, the internal temperature rise above 104°–105.8° Fahr., then we have the possibility of complication with sepsis.

Course.—The duration of this disease is somewhat brief. Its course is very acute, and the result may be decided in from 12 to 18 hours. In cases of recovery a very noticeable improvement mostly shows after from two or three days. The animals begin to stir, stand up again, appetite and milk-secretion are both renewed as well as the evacuation of dung and urine, bodily temperature becomes more equal, the pulse stronger, and the fallen temperature once more rises. Complete cure usually follows in from two to five days from the beginning of illness. Rarely does a permanent paresis of the hind legs, or a monoplegic paralysis of single extremities remain behind. If, on the contrary, the result be unfavourable, the paralysis passes gradually to the muscles of the heart and to the brain, when death follows with general muscular spasms and restlessness, not unfrequently after sudden diarrhœa, in from one to three days. Apoplectiform death is sometimes witnessed after the malady has run only a very short time. Owing to the paralysis of the upper pharynx and consequent swallowing the wrong way when administering drugs, the complaint is often complicated with pneumonia due to foreign bodies, from which death ensues sooner or later, mostly in from four to six days. Among the after-effects and complications which are occasionally met with are indigestion; necrosis of the muscles of the thigh, with pronounced œdema of the thighs (thrombosis); necrosis of the hoofs and teats; mastitis; permanent paralysis of one of the legs; contusion of the patella muscles and paralysis of the optic nerves.

Prognosis.—Under the older methods of treatment, the mortality from post-partum paresis (milk-fever) was, on the average, 40–50 per cent., but varied considerably in different times and places, according to the course of the disease. Hering and Franck estimate the mortality at 40 per cent.; Köhne gives 25-50 per cent.; Saint Cyr and Violet, Stockfleth and Ehrhardt, 50 per cent.; de Bruin, 50–66 per cent.; the Bavarian veterinary authorities, 48 per cent. Another 10 per cent. must be added to these figures to cover the mortality from pneumonia among those which recover. The introduction of the iodide of potassium treatment, especially in combination with the air method (see

p. 384), has been followed by a marked reduction in the mortality, the number of deaths being now 20 per cent. or less. The prognosis is rendered unfavourable by the following symptoms: the sudden appearance of marked signs of paralysis immediately after delivery; complete anæsthesia of the cornea; paralysis of the soft palate; and insensibility to electric stimulus.

Differential Diagnosis.—As milk fever arises in direct connection with the act of delivery, it can only be confounded with a few complaints occurring in the same connection. In this manner the so-called "paralysis," before and after parturition, becomes especially important, as during the course of puerperal fever it may occur simultaneously. This partial paralysis consists in a weakness of the back which appears several weeks before the birth, cannot be directly removed, but generally disappears on the conclusion of delivery. Or else transient paralytic signs develop in the hind legs after the birth, in consequence of stretching or contusion of the sacral plexus and other nerves, which, however, soon pass away. In both cases the general well-being of the animal is in no way disturbed. With the symptoms of brain-depression which appear in the later course of septicæmia-puerperalis, and which generally precede its fatal end, paresis of parturition cannot well be confounded.

Therapeutics.—Prophylaxis is of the utmost importance in post-partum paresis. It should, if possible, include daily exercise of the pregnant animal and decrease of her fodder ration. A gentle laxative (Glauber's salt) is also to be recommended immediately before delivery. It is advisable to milk the cow a few days before parturition. Care must be taken to ensure good ventilation, temperate warmth and prevent cold. It is best to bring the animal into a separate byre or stable, except in the case of pasturing animals, which should calve in the open (de Bruin). In stables where this disease has frequently caused loss it is advisable to wash out the uterus with a ½ to 1 per cent. solution of creolin, as a precautionary measure to prevent development of toxic matter. Trasbot considers that blood-letting, some four or five days before calving, is an effective prophylactic.

The best method of combating the disease is undoubtedly by the iodide of potassium treatment, introduced by Schmidt-Kolding (Denmark) in 1897, who assumes that post-partum

paresis (milk-fever) is an auto-intoxication caused by the agency of a toxin which forms in the udder during the development of the colostrum. He, therefore, introduces a solution of iodide of potassium (⅔ oz. in 1¾ pints distilled water) under pressure into all four ampullæ and distributes the injection by means of massage over every part of the udder. One thousand seven hundred and forty-four sick cows were treated by this method in Denmark in the years 1897–1899, and of these 1,446, that is 83 per cent., recovered (Jensen), *i.e.*, the mortality was only 17 per cent., while it had been estimated previously at between 40 and 50 per cent. More than half the cows were able to stand after 1–12 hours, while in three-fourths of the cases they could do so at the end of 18 hours. Schmidt's figures have been largely confirmed in Germany. Nevermann gives the clinical histories of 358 cases treated by 41 different veterinary surgeons, the mortality being 17 per cent., *i.e.*, 296 cows (83 per cent.) recovered. According to Eiler (Schleswig-Holstein), out of 803 cows treated, 77 per cent. were cured. Haas reports on 172 cases, treated by 41 veterinary surgeons in Austria; of these 25 per cent. ended fatally. Rusterholz describes the results obtained by 35 veterinary surgeons in 197 cases in Switzerland. The mortality was here only 22 per cent., and nine-tenths of the cows treated were able to stand after 12 hours. Of 166 cows treated in America, 72 per cent. were cured (Reep). Iodide of potassium is also recommended as a prophylactic in post-partum paresis (Kragerud). Less satisfactory results are reported by Kaiser, Meyer, Möbius, Rost, Liess, Thomann, Krämer, and others. Inflammatory conditions of the udder sometimes result from the injections and in isolated cases death from apoplexy has been observed. It has been suggested that inflammatory complications would be avoided by administering the iodide of potassium intravenously (Peter, Wessel, and others).

In place of the simple iodide of potassium injections, Schmidt-Kolbing has recently (1902) advocated their being given in combination with inflation of the udder. He first of all injects about ½ pint 1 per cent. solution of iodide of potassium into each ampulla. He then pumps air into the udder by means of an air-pump. Other authorities consider the iodide of potassium superfluous and confine the treatment to the introduction of water, salt and water, air or oxygen, into the udder. The pumping-in of air appears to be attended by results as favourable as those which followed the iodide of potassium treatment,

if not, indeed, more so. According to a Danish account, out of 914 cows treated by air-injections only, 97 per cent. recovered within a period averaging 6 hours. It is usual to employ a soft rubber apparatus (air-catheter), in which the air is filtered by passing through cotton wool (air-filter). The manner in which the air, and more especially the oxygen in the air, acts within the udder is, as yet, only imperfectly understood (oxydisation of the toxins ? mechanical action upon the distended epithelial cells ? compression of the blood-vessels ? reflex action ?).

A practical method of treatment, recommended by many veterinary surgeons, is the repeated milking of the udder until dry (removal of toxins from the udder).

For general treatment, it should be borne in mind that the administration of drugs by the mouth is attended by grave dangers. The drugs should, therefore, be confined to those absolutely essential and given with the greatest caution, as they are liable to get into the air passages and thus set up pneumonia, and wherever possible, the sub-cutaneous method should be employed. The symptoms which, above all others, demand energetic treatment are : paralysis of the striated muscles of the body ; paralysis of the unstriated muscles of the colon and bladder ; and impaired cerebral activity.

For the paralytic symptoms and general weakness the use of stimulants is commended, and by preference such as can be applied subcutaneously. Here, first of all, comes veratrin (1½ to 3 grains), caffeïn (1¼ drams), ether (hourly, ½ to 1 dram), strychnine (¼ to ¾ grain per dose). Also, to stimulate bowel peristalsis, use physostigmine (1½ to 5 grains per dose, dissolved in 30 to 75 minims of water). But physostigmine, as also the other remedies, is often unreliable. Internal stimulating remedies are also useful: wine, brandy, dilute alcohol are admirable analeptics, as are also coffee and liquor ammonii. These remedies should be given either by a stomach tube or as draughts, and not as drenches. The commonest and most easily applied external stimulants are warm vinegar, oil of turpentine, spirits of camphor, mustard oil, croton oil, diluted spirits of ammonia, or inunction of ointment of tartar emetic (1 : 4), alternate warm and cold packings and fomentations, cold douches upon the head, hot flat-irons passed along the back upon an intervening blanket. Electricity is also said to have been applied with good effect. On the advantages of phlebotomy we have formed no definite opinion.

To combat the suppressed activity of the bowels various

purgatives are used. Here comes first, physostigmine (0 : 1–0 : 2), pilocarpine (0 : 3), tartar emetic (2½ to 4 drams per dose), croton oil (20 to 25 drops per dose in mucous decoctions), aloes (1½ to 2 ozs. per dose), Glauber's salts (16 to 32 ozs.). Peristaltic action is also assisted by repeated clysters and frequent exploration of the rectum, to remove all accumulated fæces. The urine should be drawn off from the bladder. In Holland great value is attached to keeping the animal in a level position. It should lie with the fore-legs doubled under at the knees and slightly upon the right side, this position exerting a favourable influence upon the tympanites (de Bruin).

To subdue any spasms or great excitement use morphia injections, clysters of chloral hydrate and bromide of potash, inhalations of chloroform, etc. Draughts of so-called anti-spasmodic decoctions (camomile, valerian, fennel, aniseed or caraway seed) are unsafe. In general, we recommend as long a delay before slaughtering as possible.

EXCESSIVE SEXUAL IMPULSE (*Nymphomania. Satyriasis*).

Occurrence.—Excessive sexual impulse in domestic animals is described under several names, such as wantonness, ruttishness, roaring sickness, seminal or maternal staggers and also—but erroneously—as "hysteria." It is most frequent in cattle, rarer in horses, sheep and dogs. Other domestic animals, such as swine and goats, develop the condition more rarely. This wantonness is seen most in cows, mares, male dogs and rams; other male animals, such as stallions, bulls and goats, are only occasionally affected. The reason why cows suffer from excessive sexual desire lies in their lack of natural exercise, being accustomed to conceive at certain times, as well as in diseases of the genital organs caused by frequent parturition, in tuberculosis, etc. Nymphomania, as it is called in females, has been noticed to be more marked in some breeds of cows than in others. The term is applied only to abnormal excess of impulse, and no strict line can be drawn between a physiological and a pathological excess.

Etiology.—This does not constitute an independent disease, but is only a symptom arising from various causes.

1. In cows the older ones are most disposed to it; also fattened kine, as well as those, four or five years old, that have been kept too fat. Certain breeds of cattle of poor physical

development, such as the cattle of the Wester Wald (Hesse-Nassau), appear to possess a predisposition to nymphomania (Luther). In cows it has been often attributed to gastric disturbance, or chronic gastric catarrh, which yields to suitable treatment (Rychner, Giovanoli Soglio).

2. In male animals, among whom it is called Satyriasis, the disease arises chiefly from too rich feeding, especially with rye, barley and leguminous foods, which are, therefore, termed "heating-foods." Lack of work among stabled animals, especially in hot climates, also conduces thereto.

3. Nymphomania is often produced in cows by pathological changes in the generative organs, particularly by inflammation, cystoid degeneration, enlargement, dropsy, or by cancerous, sarcomatous and tuberculous new growths of the ovaries. The frequent occurrence of a morbidly increased sexual impulse as a sequel to tuberculosis led to this being at one time called "French-sickness." Tuberculosis, among these pathological changes of the ovaries, is not the chief cause, but the cystoid degeneration. Thus Schmidt found no tuberculosis in ten ruttish cows which he dissected. Tuberculosis causes wantonness only when it attacks the genitals, and, above all, the ovaries. It is wrong to imagine that the pathological changes in the ovaries must always be combined with nymphomania. The latter is, however, often found apart from any affection of the ovaries; thus, Luther examined a number of ruttish cows of the Westerwald breed, without ever finding a pathological condition of the ovaries. Especially may nymphomania be caused by diseases of the uterus which prevent conception, by chronic vaginitis and endometritis, occlusion of the os uteri, new growths and displacements, atrophy and hypertrophy of the womb.

4. Among stallions, those which suffer most from excessive sexual desire are the monorchids and cryptorchids, in whom one or both testicles are retained in the inguinal canal or in the abdominal cavity.

5. Frequent sexual excitement, such as that produced in stallions by the vicinity of ruttish mares, or the constant inflaming of mares by stallions without gratification of their sexual desire (ending in the latter procuring self-gratification by onanism), finally leads to either permanent satyriasis or nymphomania.

6. Immoderate sexuality is also observed during the outbreak of vesicles on the genitals of horses and cattle (pox), during maladie-de-coit in horses and also during rabies in dogs,

in Borna sickness in horses and in certain affections of the spinal cord.

Hysteria.—The comparison formerly made between nymphomania of cattle and hysteria in woman is unjustifiable. Hysteria in man is a neurosis of the entire nervous system, including, especially, that of the brain, which often has no reference whatever to the generative organs. Even when it is caused by certain changes in the womb (catarrh, displacement, new growth, etc.), it is rarely accompanied by symptoms of increased sexual desire. Hysteria manifests itself rather in melancholy illusions, hyperæsthesia, spasms, anæsthesia, paralysis, etc.; Hygyer observed hysteria in animals.

Symptoms.—(*a*) *In Cattle.* Nymphomania begins in cows as follows:—Apparently healthy animals become lustful at the usual period several times, but without conception, or their ruttishness breaks out at first every three weeks, later becomes still more frequent, and is constant. The cows show persistent excitement and disquiet, with a continual desire to gratify their abnormal lustfulness. They try to approach other cows and to leap upon them and ride them, and will even ride upon bulls. The strength of these impulses varies according to the individual; when very marked the signs are called "roaring sickness." This latter form is more prevalent in some districts than in others. The affected cows manifest great restlessness, have a glaring look and glittering eye, are easily startled, fond of leaping upon other cows, rub their horns on posts and troughs and dig them into the earth, sometimes even in a kneeling posture. They also paw the ground like bulls, uttering at the same time a peculiar bull-like roar, which, beginning as a gentle lowing, rises to a loud, prolonged roaring noise, which is heard day and night, without any apparent cause, but oftener in the morning, or when any stranger enters the byre. Sometimes this behaviour proceeds to perfect savageness; the beasts seem frantic if any stranger approach, even snapping their chains, rushing furiously about and attacking men. Other signs which must be noted are: considerable falling off in the supply of milk, as well as its coagulation when boiled; also a sinking of the body on both sides of the tail, as in highly pregnant animals. This is caused by relaxation of the sacro-coccygeal ligaments, and makes the tail look as though inserted higher up. We note a gradually developing bull-like nature, whereby the muscles of the neck grow thicker; and finally, an increasing emaciation and feebleness, which may at last change to cachexia.

(*b*) *In Mares.*—Here also we notice at first an unusually extreme or oft-repeated ruttishness, which may not unfrequently become permanent, and in which the animal, though duly covered, does not conceive, or else regularly miscarries. She then appears very restless, of unsteady eye, neighs often, shows great desire to urinate and strains to do so, emitting only a small quantity of urine mixed with mucus in the form of a thick, whitish-yellow substance, which she squirts out with much force by rapidly turning over the labia pudendi and pressing forward the clitoris (so-called, flashing). She is, moreover, very ticklish and excitable. Every touch on the flanks, belly or hindquarters by the hand, halter, reins, traces, or the legs and spurs of her rider causes squeaking, flashing, urination (in drops), lashing of the tail, counter-pressure, lowering of the hindquarters and even kicking and biting, so that she becomes dangerous to handle. The course of this disease is variable, may be marked by temporary loss of appetite and gradual emaciation, and may at times develop into positive fits of spasms and maniacal symptoms. Thus Eletti noticed in a nympho maniacal mare pronounced signs of madness combined with tetanic spasms, grinding of teeth, difficulty of swallowing, contraction of the belly-muscles and palpitation, a condition similar to that of hysteria in man.

Not unfrequently a considerable dulness of the sensorium shows itself later, a condition resembling sleepy staggers, and to which the name of maternal staggers is usually given. At the same time a periodical increase of these morbid symptoms is observed, corresponding with the occurrence of ruttishness. Although the signs just described will in some cases disappear for a time after the animal has been covered, yet they generally soon return, and produce, by their prolonged excitement, a very prejudicial effect upon the mare's general health.

(*c*) *In Male Animals.*—Stimulated sexual instinct, the satyriasis of horses, manifests itself in frequent erection of the penis, which sometimes becomes persistent (priapism, chorda venerea), constant unrest and excitement, continual whinnying or roaring, great excitement at sight of a mare and repeated attempts to leap upon her. If this sexual desire be not gratified, the animal's condition will sometimes rise to perfect mania. It is said that bulls and stallions, when in this state, will pursue men, and especially women (Spinola). Later the animal becomes dull, refuses food, wastes away, and, according to several observers, develops paralytic signs in the spinal medulla, result-

ing in a staggering gait and a gradually increasing cachexia, which ends in death. In stallions sometimes, though not often, a condition resembling staggers appears, the so-called "seminal staggers," to which, according to Roll, stallions which have once been used for breeding purposes and then afterwards disused are specially pre-disposed. But the title "seminal staggers" is evidently applied in literature in various senses. Some understand thereby the signs of mania which appear in the course of satyriasis, and others a persistent condition of depression of the brain occurring during the same disease, which forensically should be compared to sleepy staggers. Lastly, it seems that in some cases the viciousness developed during satyriasis has been erroneously termed seminal staggers.

Satyriasis leads to attempts at self-gratification of the sexual impulse.

Onanism.—Apart from satyriasis, the habit of self-gratification, or voluntary loss of semen, is not uncommon among male domestic animals. This cannot be regarded as an outcome of abnormally stimulated sexual impulse, because stallions and bulls which are addicted to it remain perfectly indifferent to mares and cows. The practice is commonest in dogs and rams, but is not rare among stallions and bulls. The consequences vary much according to the degree of indulgence. In some cases the disturbances are scarcely noticeable; in others we observe gradually increasing symptoms of laziness, slight sweatings and fatigue, emaciation; dulness of the sensorium; weakness and signs of paralysis, arising from the spinal medulla; enfeebled power of procreation, amounting in time to complete impotence; cachexia, epilepsy and fits resembling apoplexy. In a stallion Spinola found the lumbar medulla softened for a distance of three inches. Treatment consists in punishment, hard work, short rations and admission to coition. If these fail, castration is the only remedy. Prangé cured the evil in a stallion by making it stand two hours a day for ten consecutive days in cold running water.

Therapeutics.—The most favourable cases of nymphomania and satyriasis are those which arise from too little work and too much food. We should give more work and less fodder, separate the sexes and, if convenient, gratify their sexual desires. Besides these, purging by way of the intestinal canal should be attempted, using Glauber salt for cattle, and aloes or tartar emetic for horses. We might have recourse even to blood-letting. Cold washings and cold baths should also be applied. The sexual impulse may also be diminished by means of drugs, the best of which are chloral hydrate (horses and cattle 5 to 12½ drams in their drink); bromide of potash, bromide of soda,

sulphonal (3 ozs. daily, for horses); also sub-cutaneous injection of morphia (3 to 7 grains for both horses and cattle).

If these remedies prove unavailing, there is nothing left but castration, unless perhaps we try the suggestion of Eloire, and introduce some foreign object, such as a leaden ball, into the uterus; or follow Schünhoff's plan of widening the uterine orifice. The only, though not always reliable, cure for nymphomania and satyriasis is castration. Ovariotomy often fails, especially with mares, for Hobday operated on 30 cases, 17 of which only were cured (57 per cent.). In male animals and cows it can be performed without danger. The operation should be undertaken only when the mares have been proved to be otherwise useless, as there is risk of fatal peritonitis. But according to Cadiot, Schwendimann, Hobday, etc., carried out from the vagina, the operation is almost free from danger, even in mares.

In cases of cystic degeneration of the ovaries in cattle and horses, some authorities recommend that these cysts be crushed by the hand from the rectum (Zangger, Utz, Brüller and others). Utz says that of 100 cows he thus cured 90. Even after castration and crushing of the cysts, signs of restlessness and excitement may remain, especially if there be a simultaneous affection of the uterus, as in tuberculosis. In the case of cattle it is best, under such instances, to slaughter them without delay, even though in poor condition. For the same reason, it is not advisable to waste time over the administration of internal remedies to cattle. Parascandolo is said to have cured vaginismus in a dog by resection of the pudic nerve.

ABNORMALLY DIMINISHED SEXUAL IMPULSE.

Occurrence.—This is a pathological appearance of great importance to all cattle breeders. It occurs mainly among females, and especially in cows and female yearlings, but also in the males. Sexual impulse may be either abnormally diminished in various degrees, or absent. At the customary period, sexual impulse is absent, rare, or exceedingly feeble, lasting but a short time. If such lack of impulse be not merely transient, as is usually the case, but permanent, it amounts then in its results to sterility or unfruitfulness.

Etiology.—Among the causes of diminished sexual impulse the following are the most important :—

1. *Diseased Condition of the Sexual Organs*, viz., catarrh of the vagina and uterus (fluor albus), retention of the after-birth, hydrometra, pyometra, degeneration of the ovaries and testicles, faulty development or malformation of the same.

2. *Weakness of the Sexual Organs*, consequent upon using the male or female animal too early for breeding, excessive copulation, misuse of stimulants, onanism.

3. *Torpidity of Sexual Life*, caused by a phlegmatic temperament, and great proneness to obesity. These two usually go together, and, in cows especially, a too rich and abundant diet, combined with lack of exercise, is a frequent contributing cause.

4. *General Condition of Weakness* after a prolonged course of spare and poor diet, over-exertion in work, recent severe illness, affections of the spinal medulla ; also acute feverish, as well as chronic, constitutional maladies.

5. *Psychic Disinclination* of the male to certain female animals, and especially to those of particular colours ; *laziness* in the male. There are also fastidious stallions which prefer covering maiden animals or gelded mares, and despise those that have foaled.

Treatment.—This varies according to the originating source. Fodder and keep must be dietetically regulated. With enfeebled animals nitrogenous foods are necessary (oats, leguminous vegetables, rye, bread, raw eggs) ; and for fat ones, lessened fodder and abundant exercise. Stallions must be freely ridden or exercised, and the use of a shield may be necessary. Bulls also, if early accustomed to it, may be put to labour, or placed in a paddock by themselves. Finally, the regulation of sexual life must not be neglected. Avoid allowing excessive or too early coition to the males, or too often bringing together phlegmatic females with male animals, or taking mares frequently to the stallion. Sexual impulse may also be stimulated by administering exciting remedies (aphrodisiacs). Among these cantharides stands first and, like all similar remedies, acts best when we have merely to deal with an abnormally enfeebled impulse, and should therefore be given only at that time. To large cows give daily, for several consecutive days, 30–75 grains of powdered cantharides between two slices of bread or in wine, and to horses 15–30. Of the tincture of cantharides give cows 5 drams, horses 2½ and dogs 3–15 grains. For the same purpose all spicy, balsamic, resinous and alcoholic remedies, or such as contain ethereal oil, are useful, viz., pepper (2½–4 drams for

cows), ginger, caraway seeds in wine, mustard-seed, juniper berries, aloes, savin tops, brandy, egg-flip, alcohol, etc. All these substances produce hyperæmia of the abdominal organs, turgescence of the ovaries and testicles, with consequent increase of sexual impulse. If, however, the causes lie in organic changes of the generative apparatus, treatment is of no avail.

IMPOTENCE AND STERILITY.

I.—IMPOTENCE IN MALE ANIMALS.

This impotence, or incapacity to propagate, may arise, from inability of the male to perform the act of coition as such in correct physiological manner, in spite of the normal operation of the sexual glands; or, from incapacity to copulate fruitfully, although the accessory genital apparatus may perform its functions properly. For better understanding of the conditions we must differentiate, therefore, between "impotence of coition" and "impotence of procreation."

1. *Impotence of coition* may also be termed "relative" impotence, viz., such as can be removed. It arises from disturbances which render preparation for the act of copulation or its due performance difficult, or even impossible. The cause in such cases lies in accidental external conditions. Impulse may be strong, and the semen fruitful; but weakness may be present in the genital apparatus (compare the paragraph on the causes of diminished sexual impulse), due to disturbed innervation consequent upon chronic affection of the spinal cord and brain. Probably the term "impotence" is best applied to such conditions of weakness. The animal cannot erect its penis nor introduce it into the vagina, or discharges before the erection is complete, or sometimes the erection is so brief that discharge occurs before entrance to the vagina. Other causes may be morbid conditions of the penis, *e.g.*, kinking, phimosis, paraphimosis, new growths on it or abnormal size. Also motor disturbances, which prevent the male from leaping on the female, or render this impossible, such as painful affections of the joints, like spavin or ring-bone, or weakness in the hind-quarters, like the so-called broken back. Treatment which must aim first at removing these primary causes, should consist mainly of good food, electricity, operative help and surgical remedies. Compare also the treatment for lack of sexual impulse.

2. *Impotence of Procreation* arises either from deficient production of semen, so-called aspermia, or from the absence therein of spermatozoa, so-called azoöspermia. It may be that both testicles are lacking, or that they are atrophied in consequence of inflammatory and degenerative conditions, *e.g.*, after carcinomatous destruction, aplasia, hyperplasia of the testicles, etc. There are cases in which, in spite of apparent normal soundness of all the generative organs and especially of actual ejaculatio seminis, copulation remains unfruitful. Here it is the spermatozoa which are absent from the seed, a condition noticed in old stallions and sometimes from the very first in young ones. The fact may be established by microscopical examination of the semen discharged. Cases also occur in which the spermatozoa are present, but in a motionless, that is, a lifeless condition. The cause of this is to be sought in chronic inflammatory or degenerative conditions of the seminal canals, or in congenital aplasia of the same. Treatment for impotence of procreation is useless.

II.—STERILITY, OR UNFRUITFULNESS, IN FEMALE ANIMALS.

This may consist in incapacity for the act of coition, or for fertilization or germ-formation; and may be either transient (curable) or permanent (incurable).

1. The transitory form of sterility may arise from nymphomania, diminished sexual impulse, occlusion of the os uteri, or neck of the womb, by chronic inflammatory processes or scarred strictures. In cows and goats more especially, stenoses of the vagina, a misplacement or deviation in position or direction of the entrance of the vagina, displacement (kinking, twisting, inversion, prolapse) of the womb and new growths therein, may all produce this result; also chronic inflammatory processes of the mucous membrane of the vagina, uterus, tubuli, especially chronic endometritis (fluor albus) as a complication of parturition or of epizoötic abortion; and the abnormal retention of large yellow bodies in the ovaries which prevent normal rut. A breaking out of blisters or vesicles may also leave sterility behind. The *treatment* of all such conditions is purely surgical. It is especially easy to remove the yellow bodies, by means of pressure from the rectum, according to Swiss and Danish authorities. In the case of sterile mares, Grabensee also recommends the injection of a ½ per cent. solution of bi-carbonate contained into the vagina, with the object of neutralizing the

excessive acidity of the vaginal mucous, to which the sterilization of the spermatozoa is due. Garrett conforms the favourable effects of this treatment. The injection of bi-carbonate of soda or other alkaline solutions (soda, soap, carbolic soda, creolin, lysol, liquor cresoli saponatus), into the vagina, has been attended by equal success in sterile cows (Brandt, Handschuh, Reindl, Diem). Other methods are the stretching of the neck of the uterus and artificial insemination (introduction of spermatozoa). In one case of peritoneal rupture and permanent inflammation of the vagina in a mare, sterility was cured by stitching the ruptured peritoneum (Bournay).

2. Incurable sterility can generally be traced to absence of the ovaries, or their atrophied condition. Among such must be named: aplasia, hyperplasia, atrophy of the ovaries, cystic, carcinomatous degeneration, occlusion of the ovarian cavity (in mares) by circumscribed peritonitis, which, by forming a deposit of connective tissue, hinders the eggs from leaving the ovary; further, defects in the uterus itself, aplasia and atrophy of the same, hermaphroditism in twin-births, etc. After chronic endometritis incurable sterility may remain. Eloire produced artificial sterility in cows by introducing foreign objects into the womb, such as lead-balls and stones.

Trichomonas vaginalis. Mazzanti is incorrect in believing that the infusoria, trichomonas utero-vaginalis, which sometimes occur in the mucous secretions of the vagina and uterus in young cattle, have any connection with sterility. In man also, they occur as an accidental finding without pathogenic significance.

MILK ABNORMALITIES.

General Remarks.—These abnormalities play an important part in the keeping of cows, and as the veterinary surgeon is often consulted about them, must be treated in detail. Our present information respecting milk abnormalities we owe largely to the researches of Fuchs, Haubner, Fürstenberg and others. The causes of the various abnormalities are in general as follows:—1. Food which is abnormal either in quality or quantity. The fact that such conditions occur most frequently in cattle is accounted for by their somewhat unnatural mode of feeding. 2. Gastro-intestinal catarrh, cachectic and other morbid bodily conditions. 3. Diseases of the udder. 4. Infectious (bacterial), thermic, chemical and electrical influences

acting from without. Apart from their veterinary aspect, milk abnormalities are important owing to their secondary action in provoking disease in man and other animals.

Sub-division.—Milk abnormalities may be variously classified according to different points of view.

1st. Those which occur as *anomalies of secretion* : agalactia, milk which is too watery or too fatty.

2nd. Abnormalities caused by *external influences*, especially the invasion of bacteria ; curdling milk, that which cannot be turned into butter, slimy, putrescent, soapy, blue, red and yellow milk.

3rd. *Milk abnormalities produced by foreign admixtures*, *e.g.*, dirt, bitter and rancid substances, pigments, poisons, drugs, diseased matter and blood.

I. AGALACTIA. ABSENCE OF MILK. MILK STOPPAGE

Etiology.—Arrest of milk is one of the earliest symptoms of internal disease, and especially of stomach or bowel complaints, and arises from bad feeding, prolonged hunger, and over-exertion in work. Certain anomalies of the udder must likewise be considered, viz., mastitis, especially the chronic catarrhal form (Agalactia catarrhis contagiosa), an imperfectly developed udder, fatty udder, congestion during the time of menstruation, etc. Psychic affections, such as pining for their young and for the accustomed stall, or fear occasioned by being milked by strangers, may all affect the secretion of milk. Agalactia is met with also in those bearing young for the first time, particularly mares. The bad habit contracted by some of sucking their own milk, or its being sucked by a neighbouring animal cannot strictly be reckoned as agalactia. In a few solitary cases the absence of milk cannot be traced to any cause. Atropine is said (?) to reduce the yield ; so also do the salts of iodine and external rubbings of rosemary oil and opodeldoc.

Therapeutics.—The so-called "milk driving" remedies (galactogogues) have always been recommended for the treatment of agalactia, together with a supply of good food as fluid in character as possible, such as scalded food, brewers' grains, etc. The favourite remedies are preparations of antimony (black and yellow antimony), sulphur, chlorate of potash, fennel,

juniper berries, caraway seed, aniseed, dill, common salt, etc. These may either be given alone or in a great variety of combinations. As a so-called "milk-powder" we generally use the following mixture :—Black sulphide of antimony, 3 ounces; sulphur, 1½ ounces; fennel, caraway and juniper berries, of each 5 ounces; common salt, one pound. One tablespoonful of this mixture to be given to the cow with each feed.

Unusual Lactation.—In opposition to agalactia, secretion of milk and swelling of mammary glands sometimes occurs in many animals in the absence of pregnancy. In single cases it has been observed to arise from the animal having fancied itself pregnant, or that it had given birth. Thus in bitches (pugs) we have often noticed lactation quite independent of either pregnancy or parturition. Among 70,000 dogs treated in the Berlin dog hospital, 65 cases of this unusual lactation were observed. The same occurs also in mares, cows, she-goats, ewes and sows, deprived of the power of generation (Albrecht, Krüger, Hess, Pusch, Heinke and others). Abnormal milk-secretion has also been observed in young fillies and cow-calves (Slivnik).

II.—WATERY MILK.

Etiology and Treatment.—Watery milk is distinguished by an absence of fat and caseïn, and an excess of water. It is bluer in colour and of relatively higher specific gravity. It is caused by poor, watery food, as, for instance, an excessive or exclusive diet of mashes, turnips, turnip-tops, etc.; by disorders of stomach or bowels; by cachectic and hydræmic conditions; and, at times, by some peculiar characteristic of the breed, apart from any of the foregoing factors. *Treatment* consists in change of food, supply of dry fodder and administration of stomachics, such as common salt, 6 ounces; caraway seeds, gentian and calumba, of each 1½ ounces. Of this mixture one tablespoonful should be given with each feed. Any morbid affections of the intestinal canal, blood, etc., which may be present, must previously be removed.

III.—FATTY MILK.

Etiology and Treatment.—This is found most frequently in sheep after too rich feeding, especially with leguminous food, and also during the rutting season. The result of drinking this excessively fatty milk is to cause gastro-intestinal catarrh, diarrhœa, etc., in the lambs. The remedy lies in lighter provender for the ewes, avoidance of food which is too nitrogenous and in abundant exercise.

IV.—CURDLING MILK.

Etiology.—Premature coagulation of milk—the so called "curdling," "sweet curdling" or "cheesing" of milk—is one of the commonest faults in cows' milk, and frequently occurs also in that of goats. Its causes are varied, and may arise from digestive disorders, sour food-stuffs, such as sour mash; from diseases of the udder, especially inflammation, from hyperæmia and swelling in advanced pregnancy; nymphomaniacal conditions; over-heating of the body by movement or work. According to Fleischmann, fodder infested with uredineæ will cause milk to curdle. Of external conditions the most provocative are: great summer heat; electric conditions of the atmosphere, as during thunder-storms; ill-ventilated byres; dirty utensils and straining-cloths (especially on dairy-farms). According to Ritz, milk will curdle if yeast is kept in the dairy. Hohl has succeeded in cultivating a micro-organism (carphococcus pitnitoparus) which is found in straw and which causes milk to curdle in 24 hours.

Phenomena.—These are that the milk curdles of itself within a few hours of milking, and especially that it coagulates in boiling. The evening milk shows greatest liability to this fault. The formation of acid is only very slight—hence the name "sweet curdling"—the amount of cream is so small, that it is difficult to churn the milk into butter. In mastitis the milk when drawn from the cow, is already charged with flaky coagulæ.

Therapeutics.—Treatment is regulated by the causes. Good ventilation and coolness in the byre, as well as perfect cleanliness of utensils and straining cloths, are imperatively necessary. Immediate cooling of the milk in refrigerators is to be recommended, and also the addition of alkali, as much bicarbonate of soda as will rest on the point of a knife in each quart, or of salicylic acid (4 grains per quart). The internal administration of alkalies, especially soda, is also found useful; on the other hand, we cannot commend the use of acids internally, as some authorities have done. As a remedy for curdling milk Harms administers fennels with sulphide of antimony (13 ozs. of each; one-sixth part morning and evening). Disease of the digestive canal, udder, etc., so far as these are the causes of curdling milk, demand special treatment.

Causes of Coagulation of Milk.—These lie in the action of a ferment discovered by Pasteur in 1857, the *lactic ferment*, which decomposes the milk-sugar into lactic acid, and thereby causes acidulation and coagulation of the milk. Lister, who has called this organism the "bacterium lactis," discovered that there are also many other micro-organisms which can produce these effects in milk. To these belong, according to recent researches: osteomyelitis coccus, staphylococcus albus, citreus, cereus albus, cereus flavus, streptococcus pyogenes, bacillus pyogenes fœtidus, erysipelas-streptococcus, the bacterium, micrococcus and sphærococcus lactis acidi, the bacterium lactis aerogenes, micrococcus ovalis, streptococcus coli gracilis and others.

V.—NON-BUTTERING MILK (FERMENTING, FROTHY MILK).

Etiology.—The causes of "non-buttering," "dumb," "frothy" or "fermenting" milk are to be sought in digestive disorders in general disturbance or in bad and in-nutritious food, like turnip-tops; also in udder-affections such as occur at the end of pregnancy; in the effects of great heat or cold; in the presence of other faults in the milk (curdling, rancid, putrid); and, lastly, in that of certain micro-organisms; pre-supposing that the faults do not lie in the method of making the butter. If the milk of a cow thus affected be mixed with that of a healthy animal, the latter milk also becomes infected. Milk which will not butter, or which ferments, is a cause of much trouble in dairies and cheese factories. According to Adametz, both bacteria and blastomyces may produce this defect. The former generate gas at the cost of the caseïn, the latter at the expense of the milk-sugar. He isolated several bacteria, as well as a yeast-like fungus, the saccharomyces lactis, and proved their fermenting properties by experiment.

Symptoms.—The cream on milk which normally coagulates only during boiling, is present in a thin layer, and cannot be turned into butter, or only with great difficulty, as it coagulates, foams, and refuses to collect. On long standing it shows small, yellow spots on the top, becomes greasy, pappy, and whatever butter has formed is crumbly and soon goes rancid.

Treatment.—In addition to the various milk remedies (fennel, antimony, caraway seed, etc.), the Haubner-Siedamgrotzky acids are very effective, viz., hydrochloric acid and acetic acid. We also recommend alum (40 grains thrice daily) and chalk (Harms) three times a day in doses of 1½ to 3 oz.

If the milk have a bitter taste, add ½ oz. chlorate of lime. Contamine commends a mixture of aniseed, antimony (5 drams each), vinegar and water (about 1 pint) for eight days running. All utensils and the byres must also be disinfected.

VI.—PUTRESCENT MILK.

Etiology.—This abnormality of milk—which is very rare—is caused by infection of the milk by putrefactive bacteria, which is usually conveyed by dirty byres, stalls and utensils. Processes of decomposition in the intestinal canal, or feeding with rotten food (rancid bone-meal, for instance), may also produce it. The infecting micro-organisms are principally bacterium termo and lineola.

Symptoms.—Over the thin layer of milk small bubbles of gas, consisting of sulphuretted and carburetted hydrogen, show themselves after three or four days, burst and leave indentations behind, in which the cream has vanished. The remaining cream gradually becomes of a dirty colour and mixed with drops of oil; its taste is bitter-sweet, foul, rancid (free fatty acids, especially butyric, valerianic, caproic, caprylic and capric acids). The milk refuses to turn into butter.

Therapeutics.—In this case prophylaxis consists in disinfection and cleansing of stalls, dairy and all utensils; in the internal administration of stomachic remedies and such as check fermentation, so far as processes of decomposition in the intestinal canal require. For this latter purpose the neutral salts, common salt, hyposulphite of soda and hyposulphite of lime should be tried.

VII.—SLIMY AND STRINGY MILK.

Etiology.—Stringy milk is undoubtedly caused by the agency of fungi; accounts vary considerably, however, as to the nature of actual causal organism. Schmidt-Mülheim found, on placing such milk under the microscope, small, round, mobile bodies, which refracted the light and were about one micro-millimetre in diameter—partly in the form of micrococci and partly of beady chains. The material of fermentation is said to be the milk-sugar. Solutions of albumen or of caseïn, when mixed with the ferment, do not produce fermentation, whereas a 1 per cent. solution of milk-sugar shows distinct

mucous fermentation. The mucous matter, when extracted by alcohol, shows by its reactions the character of a vegetable mucus (similar to that of quinces); and the milk-sugar entirely disappears at the same time. More recent investigations show that several micro-organisms possess the property of causing stringy milk. Hüppe and Loeffler have discovered and isolated the one a micrococcus, the other a specific bacillus. Ratz differentiates between slimy and stringy milk. The slimy fermentation, according to him, is not the work of a particular micro-organism, but of several, among which he fully describes a diplococcus. Adametz cultivated a bacillus which rendered sterilised milk in the highest degree stringy. Guillebeau and Freudenreich, Leichmann and others have also discovered specific bacteria.

This abnormality occurs most frequently in summer, and in dirty byres or dairies, and may be attributed to bad digestion, or feeding with rotting fodder infected with bacteria. The consumption of pinguicula ("fat-herb") is said also by Müller to make milk stringy; in Sweden this has been experimentally proved.

Symptoms.—Whereas, on the first day after milking, the milk is still thin, on the second it is thickly mucous, flows badly, draws out into threads, curdles, has but little cream, an insipid taste, is difficult to churn, and the butter obtained is greasy and of bad flavour. It often presents a white, homogeneous mass, which is so tough that it will not run out when the bowl is reversed. On microscopical examination we find, in addition to the bacteria already named, that the caseïn has separated in the form of round discs (spherical crystals). Lately, slimy milk has been differentiated from stringy. The former produces a plentiful and thick layer of cream, which makes perfect butter. The stringy milk, apart from its different consistence, forms only very little or no cream, and possesses a pronounced power of infection, a small quantity being able to taint large amounts of normal milk and to render it all stringy.

Therapeutics.—The most effectual preventive of this fault in milk is thorough disinfection of the milk chambers with sulphurous acid, hot steam, etc., and careful cleansing of all utensils. Schmidt-Mülheim says that heating the milk to 150° F. destroys the micro-organisms.

In cases of digestive disorder administer internally the proper stomachics and anti-catarrhal remedies. Haubner recommends feeding with hemp-leaves.

VIII.—SOAPY MILK.

Causes and Treatment.—By "soapy milk," is understood milk which has a soapy taste, never coagulates, however long it stands, but precipitates a slimy sediment and gives a very frothy cream, which is extremely hard to churn into butter. The causes of this milk anomaly are, according to Weigmann and Zirn, traceable to the presence of a special bacillus, the so-called bacillus saponacei. These bacilli are small motile rods, with rounded ends, measuring 1.6 micro-millimètres in length by ½ mm. broad. They liquefy gelatine and produce on it round, white colonies with a central yellow spot. When placed in milk they produce in the course of a few days a slimy condition and the characteristic soapy taste. They are supposed to be conveyed in straw and hay, both of these forms of fodder being found to contain them. *Treatment* consists in removing the straw and fodder and in washing the udder.

IX.—BLUE MILK.

Occurrence.—Blue milk is observed most frequently in spring and the height of summer, particularly during moist, warm weather and in dark dairies, where it may persistently occur, even for ten years (Steinhoff). On the other hand, this condition generally disappears in the colder seasons, or after the air has been cleared by heavy rain. At first one cow only gives blue milk, but soon all the cows in a byre are infected. Very often sick cows show a pre-disposition to the production of blue milk.

Etiology.—Blue milk is caused by a special chromogenic bacterium, the *bacterium syncyanum* (Schröter). Fuchs described it in 1841 as vibria cyanogenus, and Ehrenberg gave it the name of vibrio syncyanus. According to Hueppe's more recent investigations, it occurs in the form of small rods, from two to four micro-millimètres long by ½ thick, which are motile, have rounded ends, are colourless and multiply by division and formation of spores. In sterilised milk (in which the bacteria acidi

lactici are killed) the bacilli produce only a slaty grey, or, at most, pale blue colour, owing to the absence of these bacteria and of the sour reaction which especially causes the blueness; whereas on peptone gelatine broth, it produces a green, which gradually changes to brown, and by oxydising means may be converted into blue. On sterilised potato the colour is greyish-blue. This very motile fungus decomposes albumen into a colouring matter very much like aniline blue. Its transmission takes place through the air and probably also by flies. Whether the milk can be infected by these bacilli while still in the udder is uncertain. Milk rich in albumen, the result of feeding with fodder containing much protein, is predisposed to turn blue, as also is very alkaline milk which coagulates slowly or incompletely, and thus differs from acid milk. Haubner maintains that a sudden change of food or place of pasturage conduces to blue milk, especially if the land has been manured with gypsum, and the appearance vanishes on removal to lighter pastures. Hueppe also believes that certain methods of feeding, diseases of the udder, etc., all have a secondary influence upon the blueness of milk by delaying acidulation. It appears probable that the condition is due, not only to bacillus cyanogenus, but to other bacteria (cyanea fluorescens, according to Zangemeister).

Symptoms.—These, according to Fuchs, are as follows:—One or two days after milking, spots appear, simultaneously with acidulation and coagulation, upon the cream, at first light or greyish-blue, then changing from indigo to sky-blue. They are small and irregular, the size of a pin-head, and gradually enlarging, penetrate later the layer of cream and finally also the deeper mass of milk, even turning it all blue. The consistence of the milk also changes, owing to the modifications wrought in its albuminous and fatty constituents. The caseïn loses in firmness and becomes more fluid, and the butter turns tallowy, rancid and discoloured. The blue discolouration, which appears in the form of spots or stripes, is often surrounded by a greenish, shiny border. After several days this blue milk decomposes, and putrefactive as well as pathogenic bacteria are developed, when the whole assumes a dirty grey colour. The consumption of blue milk is injurious both to animals and men, though diverse opinions are held on this point. Mosler observed symptoms of illness in man, and Steinhoff in swine, after the con-

sumption of blue-milk. Hueppe, on the other hand, found that blue-milk excited nausea but did not provoke other harmful symptoms in the subjects experimented upon, and considers that it may be used as a food for animals.

Treatment.—Milk can be prevented from turning blue by ventilation, cleansing and disinfection of the byre, dairy and all utensils, the best disinfectant being freshly generated sulphurous acid. According to Haubner, an addition of sour buttermilk may replace any other prophylactic. In some cases a change of food is advisable. Zürn suggests also washing the udder with antiseptics to destroy the bacilli.

X.—RED MILK.

Etiology and Symptoms.—Like blue milk, this also is caused by the presence of bacteria. Several kinds of bacteria possess the property of turning milk red. So far the following are known :—1. *Bacillus prodigiosus*, discovered in 1838 by Ehrenberg, the cause of so-called "bleeding bread" and "the bleeding host." The bacilli occur in the form of very short, motile rods, which are often mistaken for cocci. When cultivated on potato at 68° Fahr., they quickly produce a blood-red covering, while, on inoculating sterilised milk on its creamy surface, red dots appear, which run together and finally dye the whole area red. No discolouration of the entire milk occurs, because the development of the bacilli depends upon the presence of oxygen and is therefore confined to the upper surface. Both at lower (43–50° Fahr.) temperatures and at higher, the cultivation is white. When dissolved in alcohol and ether, the pigment shows in the spectrum three characteristic lines of absorption (one behind D and one each before E and F). The red solution is bleached by alkalies. Although very widely disseminated, the bacillus prodigiosus only occasionally causes red milk.

2. *Bacterium lactis erythrogenes*, discovered by Hueppe in 1887, which, contrary to the preceding, stains the whole of the milk red. It occurs in the form of short rods with rounded ends 1–1.4 μ long by 0.3–0.5 μ thick, which produce a rennet-like coagulation of the milk accompanied by an alkaline reaction. The colour is formed only in the dark, and in the course of two to three weeks the entire milk becomes blood-red. The pigment is insoluble in alcohol and ether, and possesses two charac-

teristic lines of absorption between D and E and a third in the blue part of the spectrum.

3. *A kind of Sarcina* also turns milk red (brownish red), as shown by Adametz.

4. Keferstein has succeeded in obtaining a micrococcus which he considers the specific cause of red-milk. Probably there are other bacteria with the same property. Red milk should be prophylactically treated with disinfectants in the same way as blue-milk.

Yellow Milk.—This is also nearly related to blue milk. The colourless, mobile organism, *bacterium synxanthum* (Schröter) produces a pigment like aniline yellow, ranging from yolk-yellow to lemon, which sometimes appears with green and blue, and is more frequently seen in boiled milk than in unboiled. Lactic acid prevents the development of the ferment. According to Schröter, after 48 hours a yellow, spotted discolouration appears in the boiled and already coagulated milk, with simultaneous alkalinity of the upper surface, and increase of the whey-fluid and of softness, with disintegration of the milky mass into small coagulæ. After six days the milk is changed to a lemon-yellow, watery fluid, very poor in caseous flakes. There seem also to be other bacteria which produce a yellow colour in milk. In chronic, infectious, epizoötic mastitis, the milk sometimes assumes a yellow colour (*cp.* Text-book of Surgery and Obstetrics).

XI.—FOREIGN MATTER IN MILK.

Of foreign substances in milk the most important are: dirt, abnormal flavouring matter, scents, pigments, drugs and poisons, also morbid substances such as blood or pus. The various bacteria already named, as the cause of many milk abnormalities, must also be included in the list.

1. *Dirt.* This comes from the cow's udder, which is often smeared with dung, and consists, therefore, almost entirely of cow-dung. If the cow be diseased (diarrhœa, enteritis) infection may be conveyed to those drinking the milk. Uhl found, for instance, from $\frac{1}{6}$–2 grains of cow-dung per pint of milk taken in the market at Giessen. *Prophylaxis* consists in cleansing the udder prior to milking, and in supplying dry straw. No cow's milk can be considered hygienically clean but that which after standing for two hours in a vessel with transparent bottom, shows no sediment (Renk).

2. *Abnormal flavouring matter.* Such matter arises from food-stuffs consumed when in process of decomposition. We may mention the following: rancid oil-cake, bad or frozen

potatoes and turnips, sour brewers' grains, or large quantities of turnip-leaves or turnip-tops. These, according to Schumacher, contain an ætherial oil. Also ensilage and fodder that has long lain in a heap, leaves of trees, chestnuts, dandelion, wormwood, tansy, garlic, artichokes, thistles, the refuse of rape and linseed, an excess of oat or pea straw, olive oil, rape oil, linseed oil, fish refuse, etc. Dirty vessels and utensils may also cause this defect. In all these cases the milk obtains an unpleasantly rancid, bitter, sweetish, salt or harsh flavour. Jensen states, moreover, that the bacillus fœtidus lactis produces an offensive rancid smell in the milk, with an oily flavour of turnips. Less disagreeable are the ætherial oils of the aromatic drugs: aniseed, caraway, fennel, etc. The remedy lies essentially in change of food and in keeping all vessels, etc., clean. Recent investigation shows that bitterness is produced in milk by bacteria. Krüger attributes it to an organism called proteus vulgaris; Hueppe says it arises from formation of caseïn-peptone by the bacillus butyricus and the potato-bacillus. Weigmann has proved a bacillus and Conn a micrococcus to cause bitter milk, so that evidently several bacteria possess this property. In order to avoid contamination of the other milk, cows giving bitter milk should be isolated.

3. *Strange odorous matter* gets into milk after feeding on onions and garlic, or after using highly-scented drugs, such as asafœtida, camphor or turpentine. Odour is also largely absorbed from the entire environment, *e.g.*, after disinfecting a byre with carbolic acid. In one case recorded, milk became so strongly impregnated after standing in a place thus disinfected, that a dog drinking it showed signs of carbolic poisoning (Zorn). Milk will sometimes absorb the smell from a decomposing carcase, when near it.

4. Of *Pigments*, yellow, yellowish-red and blue are met with. The yellow and red come partly from certain plants, *e.g.*, carrots, rhubarb and crocus; and in part they are explained by admixture of biliary pigments (icterus), blood and pus in the milk. In certain inflammations of the udder a yolk-yellow colostrum occurs in the milk. The blue pigments—apart from so-called "blue-milk"—have their origin in certain plants, such as alkanet, horsetails, dog's mercury, buckwheat, flowering rush and plants containing indigo.

5. Among *Drugs* the following pass into the milk, or at least their chief constituents, and produce corresponding effects upon those drinking it: camphor, ether, oil of turpentine, asafœtida,

chloroform, tartar emetic, aloes, arsenic, hellebore, belladonna, henbane, stramonium, colchicum, corn-cockle, castor-oil cake, hemlock, morphia, strychnine, veratrine, euphorbium, senna leaves, salicylic acid, carbolic acid ; the various salts (Glauber, common salt, soda, potash, sulphate of magnesia, borax, etc.) ; the compounds of bismuth ; salts of lead, of copper, zinc and iron ; the compounds of mercury ; iodine. Alcohol can be tasted in the milk of cows which have been fed on the refuse from distilleries.

6. The *products of disease* are not unfrequently transmitted through milk to men and animals. The most important instance of this is the *transmission of tuberculosis in milk.* This has been demonstrated in man. Experiments in feeding with the milk of cows suffering from bovine tuberculosis (see Vol. II.) have proved its power of infection to be very great. Friis found that 14 per cent. of the milk of commerce contains the bacilli of tuberculosis, and that mixed milk does not protect from infection. According to Lehmann, the milk of tuberculous cows is also poorer in fat and albumen. That of cows suffering from *pulmonary disease* is thick like colostrum, of a peculiar flavour and soon turns putrid. Haukold saw men vomit after drinking it. In *foot-and-mouth disease,* which is often communicated to men and animals through milk, Herberger found the milk imperfectly coagulated, viscid, slimy, resembling colostrum, bad in taste and smell, poorer in sugar and caseïn and sometimes containing ammonium carbonate. In *rinderpest* the fat is much diminished in quantity, and the sugary contents almost nil. Milk is also *said to be* capable of transmitting typhus, scarlet fever, cholera and diphtheria to man.

7. *Blood,* that is to say, *hæmoglobin* in milk, can be traced to several causes. Not unfrequently blood may be noticed in the milk immediately after calving, and lasts for about fourteen days. In other cases it may be traced to mastitis, injury inflicted on the udder by a blow, rough milking or sucking, congestion of the udder during calving, irritation by substances containing turpentine, hæmoglobinæmia, a rapid change to rich, concentrated and nitrogenous foods, especially clover, etc. The phenomena consist in uniform (hæmaturia) or streaky discolouration (red) of the milk (mastitis), formation of a sediment of red coagulæ, etc. The treatment is partly local, partly general. For further details on "bloody milk" see the text-books on obstetrics ; and for details on milk-changes produced by mastitis, consult those on surgery.

Bacteria.—Certain bacteria which appear in milk are scarcely to be classed as foreign substances. Besides the acid-forming bacteria named in connection with curdling milk (the bacterium lactis Lister, in particular), the following, according to Marpmann, also occur in milk: a bacterium lactis acidi, a sphærococcus lactis acidi, a micrococcus lactis acidi and a bacterium limbatum lactis acidi. Of organisms which do not produce acidity the commonest, according to Loeffler, is the potato-bacillus (bacillus mesentericus vulgatus) then the gum-bacillus (bacillus liodermos), a bacillus of butyric acid, a white bacillus and finally the mould-fungus (oidium lactis, penicillium glaucum, and some kinds of aspergillæ). According to Happich, the inactive bacteria are the micro coccus agilis, luteus and roseus, as well as several varieties of the sarcina. Abba says that the bacillus coli is invariably present in milk. The investigations of Cnopf, Freudenreich and others have shown that in the cleanest of dairies and with the very best milk, samples taken from the large cans a few minutes after milking contain 100,000 germs per cubic centimètre. In six hours this enormous figure rose to six millions of germs per cubic centimètre! By passing the milk through a centrifugal machine it may be freed from some portion of its bacteria. Thus Wiss has shown that the scum deposited on the sides of the machine is seven times richer in bacteria than the milk itself which has been so treated. This process is therefore of importance for the purifying of milk from infectious matter, such as tubercle-bacilli (Bang, Scheuerlen, Obermüller, Ostertag).

Milk Grit.—A certain milk abnormality is so named in Switzerland, in which the otherwise normal milk shows upon the hand when milking fine, white, soft coagulæ or flakes. These arise from dried milk in the orifice of the teat, and are consequently found in the milk first drawn (Guillebeau and Hess).

Infectious Aglactia.—This infectious drying-up of the milk in sheep and goats, described by Oreste, Brusasco, Schlossleitner and others, is, moreover, complicated with lameness, blindness, etc., and evidently belongs either to the sphere of epizoötics, or of poisoning.

Cheese Defects.—There is a very complete work upon this subject, with special reference to abnormal conditions governing the ripening of cheeses by Adametz.

CHAPTER IX.

DISEASES OF THE HEART AND LARGER BLOOD VESSELS.

INFLAMMATION OF THE PERICARDIUM. PERICARDITIS.

General Observations.—This condition is very common in cattle, but rare in other domestic animals (horse or dog). Pericarditis in cattle is distinguished from the same malady in other animals by being usually traumatic in origin, and frequently complicated with inflammation of the muscles of the heart or myocarditis, as a consequence of which the whole aspect of the disease is changed. A similar traumatic pericarditis occurs also, though rarely, in goats, but in horses and other domestic animals the condition is usually idiopathic. Consequently, in discussing inflammation of the pericardium, a clear line of division must be drawn between the traumatic pericarditis of cattle and goats and the idiopathic pericarditis of the other domestic animals, on clinical, anatomical and therapeutic grounds.

I.—PERICARDITIS OF CATTLE. TRAUMATIC PERICARDITIS AND CARDITIS.

Etiology.—This is a very common disease among cattle and by far their most frequent heart-affection. The question may be asked: Is there any non-traumatic pericarditis among cattle? A priori, we must answer, yes, for the pericardium of an ox may suffer inflammation from cold, or in the course of acute muscular or articular rheumatism, in infectious diseases such as septicæmia, or in connection with parturition or pleuritis, just the same as that of a horse. All these processes have been indicated, in addition to wounds or bruises, as causes of pericarditis. It was the usual impression that most of the cases described in literature as "rheumatic" pericarditis were really

of traumatic nature. It seems to have been a general custom among observers to diagnose all cases of inflammation of the pericardium, in which dissection revealed the presence of no foreign substance in the heart, as simply of rheumatic origin, forgetting that sharp-edged objects may work their way out of the heart into the second stomach. In any case, the greatest care is needful in diagnosing rheumatic pericarditis in cattle. Somewhat more frequent—though rare as compared with the traumatic form—may be the inflammation set up in the pericardium in the course of septic metritis, but here it is generally one symptom only among many other organic disturbances. Respecting the pericarditis which occurs during bovine tuberculosis, see the chapter on "Tuberculosis." Concerning the entrance and nature of the sharp-edged foreign bodies and their penetration from the second stomach, we refer our readers to the etiology of traumatic inflammation of the stomach and diaphragm. We would here merely mention the anatomical fact that the second stomach is only a short distance from the pericardium and separated from it only by the diaphragm. This explains the frequent injury of the heart and its enclosing membrane by sharp foreign bodies. Pericarditis caused by wounds inflicted through the ribs, *e.g.*, by goring or fractured ribs, is extremely rare. In one case on record the broken blade of a fleam was found in the heart (Kolb).

Post-Mortem Conditions.—The changes in the pericardium and heart in traumatic pericarditis and carditis are usually far advanced, so that the early stages and development cannot be followed. We generally find in the pericardium an exudation of fluid or solid nature, adhesion of the heart to the pericardium, a thickening and expansion of the latter and changes in the muscles of the heart, as well as secondary changes in the lungs, diaphragm, etc.

This pericardial exudation is varied in its composition. Sometimes it is purely serous, then fibrinous, purulent, hæmorrhagic or ichorous, and its colour is accordingly sometimes pale and clear, then milky or whey-like, sometimes whitish, flaky, or greyish-yellow, yellowish-green, reddish, brown, dirty red, etc. Its formative elements also change beneath the microscope, red and white blood-corpuscles, drops of fat and bacteria being frequently present. The fluid masses are often mingled with putrid gases, which have either been generated in the pericardial sac itself or have penetrated thither from the second

stomach, and are mostly more or less foul in smell, though at times quite odourless. The quantity of this exuded matter may be even enough to fill a stable bucket (2–3 gallons). Great pressure is exerted thereby upon both the heart and its adjacent organ, the lungs, it compresses the auricles and larger veins and greatly distends the pericardium. If much prolonged, a chronic dropsy of the pericardium (hydro-pericardium) may develop. In rare cases we find blood in the pericardium, which has reached it after perforation or rupture of the cardiac walls, and which may amount to some quarts.

The solid exudate consists of fibrinous, gelatinous coagulæ, which later become organised into connective tissue. They occur in the form of deposits of filamentous, string-like proliferations between the heart and pericardium. The inflammatory coatings which overlay the heart are particularly characteristic, and present thick deposits of yellow colour (varying from that of the yolk of an egg to that of an omelette), possessing a villous, nodular and even warty surface, and make the heart look exceedingly large, and its surface as though gnawed and hacked (shaggy heart, *cor villosum*). The epicardium itself is thickened and changed into a leathery membrane, which is sometimes incrusted with salts of lime, and envelops the heart as in a rigid cover. The coagulæ lying on the heart sometimes become slimy and undergo fatty degeneration in their lower layers. The solid exudation adhering to the heart and pericardium is of stringy, ribbon-like or tendinous character, and partly inter-penetrated by newly-formed vessels. Sometimes there is a complete union of the heart and pericardium. Occasionally after retrogression of the exudation in the pericardium, so-called macula lactea are left behind, viz., indurated, cicatricial thickenings.

The heart itself shows serous infiltration, with circumscribed fatty degeneration and softening of its muscular fibres; also inter-muscular abscesses, as well as cavities lined with abscess-membrane, which represent the space formerly occupied by the foreign body or else are mere losses of substance. They are then ulcerous, dark red in colour and surrounded by a yellowish border (wounds in the heart). The foreign object is sometimes found in the lumen of a ventricle, at others it lodges in the cardiac wall, or lies in the pericardium. In one case Camoin saw a canal which ran from the left ventricle to the second stomach, through which the blood poured directly from the one into the other. In prolonged cases the heart becomes hyper-

trophied and dilated, or else, in consequence of pressure by the pericardial exudation, is atrophied, relaxed and small. The pericardium may, in course of time, become as thick as a man's thumb; it is also frequently much distended, so as to contain three gallons and more of liquid. We also observe pleuritis, adhesion and circumscribed traumatic inflammation of the lungs, adhesion of the pericardium to the diaphragm, and also—in traumatic inflammation of the stomach and diaphragm—we find certain local anatomical changes.

Symptoms.—The aspect of traumatic pericarditis forms a continuation of the phenomena described in traumatic inflammation of the stomach and diaphragm, and consist in chronic gastric disturbance without any assignable cause, with extraordinarily inconstant and changeable course, complicated with pain in breathing, in straining or in the passage of fæces, or on palpation of the diaphragm; also intestinal hæmorrhage and great emaciation. The symptoms defy all attempts at relief by drugs. With these evidences of chronic gastric catarrh, and not rarely after an apparent diminution in their force, striking characteristic heart and lung symptoms appear, accompanied by general disturbances. These usually come on rapidly, and sometimes after a previous birth or exhausting march (driving to market, etc.).

1. **Phenomena of the Heart.**—At the outbreak of the inflammatory affection the heart-beat is stronger, palpitating, jerky and even tumultuous, so as to be audible three or four yards away; sometimes a distinct back-stroke can be heard. Later, as the exudation accumulates in the pericardium, it often ceases entirely, or is very much weakened, but more diffused, feeling like the undulations of waves. Percussion of the heart, which may be very painful, as is that of the entire region of the heart, demonstrates an increase of cardiac dulness proportionate to the growth of the pericardiac exudations. We not unfrequently notice, as one of the first symptoms, a tympanitic tone spread over the region of the heart, which is of great diagnostic importance, being due in most cases to separation of the heart from the pericardium by the interposition of exuded fluid, and to accumulation of gas in the pericardial sac. This is partly generated by decomposition of the exudations therein, but comes also in part from the second stomach. Here and there the tympanitic tone extends over the entire left wall of the chest

(pneumo-thorax, pleuritis, compression and atelectasis of the lungs). Palpation of the heart by the hand reveals in dry pericarditis a distinct friction, which keeps time with the action of the heart; where exudation is present, wave-like movements can sometimes be felt.

Auscultation of the heart is further of the greatest possible pathological importance. The ear must be placed near the fifth rib, or behind the left shoulder-blade or humerus, over the triceps. It is also sometimes advisable to try also the right side. We then hear the true heart-sounds perfectly clear, although perhaps less loud and distinct owing to other cadences. Secondary sounds after the first or second heart-sound are exceptional, and indicate valvular disease and endocarditis. On the other hand, we usually hear in all cases the pericardiac sounds which are characteristic of pericarditis. These are of very different nature, according to the composition and amount of the exudation. At first, and so long as there is not much fluid in the pericardium, and the exuded matter is of comparatively dry and fibrinous composition, we perceive a decided rubbing sound, which is caused by contact of the two rough inner layers of the pericardium. With this friction sound we also gain the impression of creaking, grinding, grating, humming and stroking. In general, this rubbing noise is not often heard, as it occurs only in the early stages of plastic-exudative pericarditis. More frequent, and corresponding to the usual fluid character of the exudation, are the sounds of liquid in the pericardium. When this is moderately full, splashing noises can be heard some yards away; or again, they are like clapping, gurgling and clucking, a kind of frothy rattle; or at other times the sounds have a metallic ring, like that of a falling drop, or are quite dull and fluctuating. This latter is when the pericardium is becoming over-filled. Whereas the rubbing noises are strictly connected with the pulsations of the heart, the other sounds may either precede or follow them, and at times cease altogether for a while. They all cease on re-absorption of the exudation, and in general synchronise with the heart-beats.

2. **Phenomena of the Lungs.**—The compression of the auricles by the pericardial exudations, combined with the diminished efficiency (insufficiency) of the heart, soon produces a congestion of blood in the lungs, a passive hyperæmia of that organ, which shows itself in quickness of breathing, especially after movement, and in slight fits of coughing, and

may sometimes lead to violent action of the breathing apparatus of dyspnœic nature. The symptoms of a lung affection then become prominent, and may be mistaken for an independent pulmonary disease, such as inflammation, phthisis, or even tuberculosis. As complications of the pericarditis with traumatic pleuritis and pneumonia, caused by the same foreign body, may also arise, the diagnosis is at first liable to be erroneous, though a careful examination of the heart will soon correct any such error.

3. **Phenomena of the Jugular Vein.**—With the appearance of the pericardial sounds, that is, about 4–6 days later, and often not for 8–14 days, the jugular veins also become very highly charged, owing to the pressure exerted by the exudation upon the right side of the heart, whereby the blood in them becomes engorged, and they show—though not always—a so-called "venous pulse," *i.e.*, distinct movement synchronising with the action of the heart. Doughy, cold swellings simultaneously develop in their vicinity, which, when the head is raised, appear on the mouth only, but when it is sunk or stretched out, are found around the larynx as far as the lower jaw and cheeks, and at times extend between the forelegs to the breast and abdomen. This congestive œdema occurs also in other affections of the heart, but, when combined with the other phenomena of the heart, offers a very characteristic symptom of traumatic pericarditis, although one which is not found in every single case.

4. **General Phenomena.**—The irregularity of the heart's action is transmitted also to the pulse, which is at first very rapid (up to 120 a minute), then weak, often thready, and at times irregular. The speed varies meanwhile with the intensity of the disease. Temperature is also changeable, a circumstance in some respects important for the diagnosis of traumatic pericarditis; it may rise to 106°–107° Fahr. The temperature of the exterior also varies often, the extremities especially being first cold and then warm, while the animals shiver and tremble. In advanced cases the general health is seriously affected. The animals are very listless, appear anxious, have a pitiful look, and groan when they lie down, move, breathe, etc. The body is stiffly held, the neck poked out straight, and the elbows stand out from the sides of the breast. All movement, especially sideways, is avoided, the animals keep

getting up and lying down again, or cannot be brought to their feet at all. The visible mucous membranes are at first more highly reddened; later they become pale, cyanotic or yellow. Emaciation steadily progresses; hydræmic symptoms set in, and occasionally intestinal hæmorrhage appears, the animals perishing of exhaustion or asphyxia, after having, in some cases, also shown pleuritis in addition to pericarditis. Sometimes, towards the end of the disease, the complete aspect o pyæmia is shown, with metastasia in the inner organs (brain, spinal medulla, kidneys, liver, muscles, joints and tendon-sheaths).

The *duration* of the ailment is generally long, lasting for months, and the course very slow, interrupted by periodical exacerbations. In rare cases death follows quickly in a few days, in consequence of perforation of the heart and hæmorrhage into the pericardium. Recovery is occasionally seen, as, for instance, when the foreign body slips back into the second stomach. The time which elapses between the inflammation of the stomach and diaphragm and the pericarditis may be weeks or months, and depends upon the nature of the foreign object, how the animal is used, its feeding, the number of times it bears young (advance of the foreign object by labour pains) and accidental circumstances. Much thickening and adhesion of the pericardium point to a duration of the condition extending over some months.

Differential Diagnosis.—The diagnosis of traumatic pericarditis is not difficult, if the examination be thorough. The previous chronic digestive disturbances, abnormal activity of the heart, the appearance of characteristic pericardial heart-sounds, venous engorgement, œdematous swellings, combined with the chronic and finally fatal course (in most cases) offer safe grounds for a sound judgment. At the commencement of the condition the coincidence of three factors is of great importance in diagnosis:—1. Incurable chronic gastric catarrh; 2. tympanitic tones in the region of the heart; 3. disturbed breathing (shortness of breath, cough) in the absence of any demonstrable change in the lungs themselves. Diagnosis is difficult in tuberculosis of the pericardium, as well as in those cases where the true cardiac symptoms are absent owing to the fact that the pericarditis is detected either at its very commencement or when the condition is already on the wane. In such cases, Gmelin recommends the subcutaneous or intravenous injection of digitalin ($\frac{1}{3}$ gr.–$\frac{3}{4}$ gr.), to increase the activity

of the heart and raise the blood-pressure, and so render the cardiac sounds more readily audible (confirmed by Schmidt, of Dresden). Digitalis is contra-indicated in traumatic pericarditis, but is indicated in the other cardiac affections. According to Otto, a peculiar muscular tremor in the neighbourhood of the elbow after prolonged standing, is a reliable diagnostic symptom of traumatic pericarditis.

Therapeutics.—No time should be wasted in treating traumatic pericarditis and carditis; if its presence be positively established, have the animals promptly slaughtered. On this point all opinions concur. Only at first, and so long as there is a slight hope of improvement and no immediate danger of death, or if the diagnosis be doubtful, we may attempt to regulate the action of the heart by administering digitalis (30–75 grains per diem), caffeïn (1¼ drams), or tincture of strophanthus (2½–6 drams); or to stimulate peristalsis (tartar emetic, saline remedies), so as to remove if possible the foreign body. Such efforts are, however, usually futile. Better perhaps would be a surgical operation, consisting either in puncture of the distended pericardium, or removal of the offending object therefrom, where it may possibly be palpable or visible (Bastin). Whether laparotomy, with the opening of the rumen and the manual removal of the obstruction from the pericardium, as recommended and practised by Meyer, is successful when once the inflammatory condition has supervened, is at present unknown. Imminger states that he has obtained good results by simply lowering the hind-quarters of the animals in the stall.

II.—PERICARDITIS IN HORSES AND THE OTHER DOMESTIC ANIMALS.

Etiology.—In contrast to the pericarditis of cattle, that of horses, dogs, sheep, etc., is in the vast majority of cases idiopathic, that is, produced by inner causes and not by wounds. Frequently these are of rheumatic nature and traceable to chill, to which horses are peculiarly liable. Schindelka has often observed pericarditis in horses after a long and uninterrupted railway journey. Or else it is developed in the course of general infectious disease, such as acute muscular and articular rheumatism, or septicæmia, phthisis or pneumonia. It may also arise from the spread of inflammation from an adjacent organ, especially from endocarditis, pleuritis,

myocarditis and pneumonia, or after an empyema caused by perforation of a lung-abscess, when it would first appear as an external pericarditis. Finally, it is in horses sometimes traumatic in nature. Thus, Henniges saw a sewing-needle, which had penetrated from the pharynx of a horse into the left auricle; Lewin, in the pericardium of another, found a darning-needle, which had probably made its way from the duodenum through the diaphragm. Broken ribs and small shot may cause pericarditis in dogs. In two cases, fatal pericarditis developed in horses as a consequence of lysol poisoning, the lysol having been employed to destroy lice.

Post-Mortem Conditions.—The first pathological changes in pericarditis are injection, ecchymosis, opacity and swelling of the inner serous layer (pericarditis interna), with a deposit of soft, thin and gelatinous exudate, which forms an easily detachable film. According to the origin we may find in the pericardium a serous, fibrinous (chills), hæmorrhagic, purulent or ichorous exudation (infection, sepsis), in larger or smaller quantities (up to six gallons in a horse), from which, if the malady be prolonged, hydropericardium may develop, particularly in dogs and sheep. Later on there appear leathery, nodulated coatings upon the pericardium (*cor villosum*); adhesion of the pericardium to the heart; formation of encapsuled cavities between the heart and pericardium, and hypertrophy, or even atrophy of the heart. Prakke found the heart in one horse no larger than a child's fist, and its walls in some places no thicker than a playing-card. In other respects, the conditions are described as mainly those given in pericarditis of cattle.

Symptoms.—These show themselves in horses and the other domestic animals at first in palpitating, tumultuous beating of the heart, which later diminishes as the exudate increases; in extension of the cardiac dulness upwards and backwards; in pericardiac, rubbing sounds (friction fremitus); very frequent, small, irregular and unequal pulse; strained and even dyspnœic breathing; jugular vein pulse and rise of temperature (102° to 105° F.). At times we can perceive nothing characteristic beyond the high fever, imperceptible heart-beat and difficult breathing. *The course* is sometimes acute, when death may suddenly ensue in consequence of rupture of the heart; at other times it is chronic. In dogs we must include

among the symptoms of hydropericardium : great difficulty of breathing, general cyanosis, congestion of the jugulars, imperceptible or fluctuating, gurgling heart-beat, rapid and irregular pulse, dropsy, with great emaciation and feebleness.

Differential Diagnosis.—Pericarditis may be confused with endocarditis and pleuritis as well as with certain pulmonary affections, although upon careful examination it should be possible to recognise and exclude the latter. Pericarditis may be distinguished from endocarditis by the difference in the pericardiac sounds. This is in contrast to the endocardiac after-sounds a friction noise, which appears to arise close to the ear, and may sometimes be felt, and is not so strictly bound to the two phases of the heart's action, its systole and diastole, as are the endocardiac sounds. Moreover, we hear the heart-sounds quite normally with pericardiac sound. Less easy is the distinction between pericarditis and pleuritis, which also is accompanied by characteristic rubbing noises. This, however, synchronises more with the rhythmical breathing than with beating of the heart, and in pleuritis the peculiar heart-phenomena are absent. Since, however, an internal pericarditis often occurs with an external one, which latter is synonymous with pleuritis, and since pericarditis, moreover, sometimes occurs secondarily after pleuritis, the distinction between the two is often very difficult or even impossible. Röll advises, in doubtful cases, to decide in favour of pleuritis, rather than pericarditis, owing to its more frequent occurrence.

Therapeutics.—The prognosis of inflammation of the pericardium is much more favourable in horses, dogs, etc., than in cattle, and its treatment therefore more successful. It consists in the avoidance of every excitement and movement, and the administration of cooling and antiphlogistic remedies. Among these come at the outset the local application of an ice-bag, or permanent irrigation of the cardiac region with cold water. Horses are best placed under a water-tap. To regulate the action of the heart, administer digitalis internally in small and repeated doses (horses 30 to 75 grains, dogs 1 to 1½ grains). Calomel, tartar emetic and the neutral salts may also be used. The external application of a mustard poultice, or of spirits of mustard on the cardiac region is also useful. Where the course is slow, try purgatives or diuretics. Should the exudation reach a dangerous point or not diminish, despite

all internal remedies, do not delay to puncture the pericardium in larger animals, and withdraw the exuded matter with a trocar. For the smaller animals, use Pravaz's syringe. In horses make the incision—after first cutting the skin—on the anterior edge of the fifth or sixth rib, a hand-breadth from the lower surface of the breast; in smaller animals on the site of the dulness. To remove the danger of heart-failure or general weakness, administer stimulants: wine, camphor, coffee, alcohol, ether, etc.

Pericarditis in Pigs, Sheep and Dogs.—According to Rieck, rheumatic fibrous pericarditis occurs with considerable frequency among pigs. Among 5,160 slaughtered swine, the condition appeared in 151, that is, in 3 per cent., and in some cases it was complicated with pleuritis and peritonitis. According to Trasbôt and Anacker, an enzoötic pericarditis occurs in sheep which runs a very acute course and ends fatally after a few days. Petit describes 15 cases of tuberculous pericarditis in the dog; of these, 6 were serous, 6 hæmorrhagic, and 3 adhesive.

Hydropericardium.—Dropsy of the pericardium is an accumulation of serous transudate in that organ. It is always a secondary condition which follows general engorgement of blood after diseases, such as chronic nephritis or hydræmia. It may be clinically recognised by the disappearance of the heart-beat, enfeeblement of the heart-tones, with enlargement and diffusion of heart-dulness.

ACUTE ENDOCARDITIS.

Etiology.—Acute inflammation of the endocardium is most frequent in horses and cattle, also in dogs and pigs, but rarer in other domestic animals; for instance, we have known only one case in a cat, but it is by no means so rare as is generally thought. In most cases it is produced by infectious matter circulating in the blood. In cattle it is especially acute articular rheumatism (polyarthritis rheumatica), which not rarely becomes complicated with acute endocarditis. In pigs erysipelas occurs as a frequent cause of endocarditis. We must also name septicæmia and pyæmia, *e.g.*, in conjunction with septic metritis as well as general infectious diseases, such as foot-and-mouth. In pigs an acute articular rheumatism may even lead to death from acute endocarditis (Roth). In horses we find acute inflammation of the endocardium with anthrax, pneumonia, rheumatic laminitis, and also with severe colic. In dogs there seems to be a contagious malignant endocarditis

(Fröhner). Also, after intra-peritoneal injection of pneumococci cultures in a dog, an ulcerous endocarditis was observed (Michaëlis). Chills play an important part, with infection, in producing so-called rheumatic or idiopathic endocarditis in horses (Trasbôt). An inflammatory process may likewise spread to the endocardium from the myocardium and pericardium, and even from the lungs and pleura. Seldom is the cause traumatic. Ebinger saw a case of endocarditis in a cow produced by the blow of a horn with consequent broken rib. A predisposition to endocarditis seems to exist in old age, resulting from certain degenerative processes, such as arterial sclerosis of the valves of the aorta; or in early youth from hereditary influences (Burke saw all the pups in a litter perish of the disease); and also after recovery from a severe illness. Among drugs which, if administered too long or in too large doses, produce endocarditis, digitalis must take the first place.

Bacteria.—Wyssokowitsch has made some very interesting experimental investigations into the etiology of ulcerous endocarditis in animals. By irritation of the valves, with subsequent intra-venous injection of streptococcus pyogenes, he produced in rabbits an endocarditis resembling the ulcerous form (mycotic) in man. His experiments prove that micro-organisms which are otherwise harmless to animals may produce severe and fatal ulcerous endocarditis if the valves be injured, and that a very slight injury to the endocardium suffices for this end. He obtained similar results with staphylococcus pyogenes aureus and with coccus sepsis.—According to Dessy, every case of endocarditis is caused by micro-organisms. In addition to the pyogenic cocci, diplococcus lanceolatus capsulatus is also said to produce the disease. Jensen found the bacillus coli together with other schizomycetes in the ulcerous endocarditis of dogs, and Thomassen found the staphylococcus pyogenes and the bacillus coli in the same condition in horses.

Post-Mortem Conditions.—The macroscopic changes vary according to the degree and cause of the malady. In the slighter rheumatic forms we find small, knotty prominences, the size of a millet seed, especially on the free edges of the valves and on the tendons and papillary muscles. They are greyish-white to whitish-yellow in colour and, when present in numbers, form nodular, wart-like, lumpy protuberances, covered partly with bloody and partly with colourless and stratified fibrinous coagulæ (endocarditis verrucosa). These growths may develop on the margin of the valves to large polypous proliferations, which cause stenosis and insufficiency of the respective ostia

and valves. If the granulæ are very small, the surface of the endocardium is rough and finely granulated. The endocardium is diffusely swollen, opaque, reddened and covered with coagulæ ; and circumscribed hæmorrhagic foci are also found in the sub-endocardial and sub-epicardial tissue, as well as between the muscular fibres. The severe septic forms develop into necrosis and ulceration of the endocardium (endocarditis ulcerosa, maligna, diphtheritica, bacteritica). The ulcers are mostly local, in size from a lentil to a shilling, and are covered with dirty discoloured, friable, brittle and necrotic deposits, which are torn away by the current of blood and carried into other organs. Thus it is we find purulent metastases in the lungs, kidneys, liver, spleen, and even in the coronary arteries of the heart (abscess of the heart).

By means of the microscope we can prove that in verrucous endocarditis there is a sub-endocardial exudation with small-cellular infiltration ; and also an exudation in the ulcerous form, but with coagulation and necrosis of the tissue and transformation into granular and flaky matter, with deposits of micrococci colonies. The former leads either to re-absorption of the exudation and recovery ; or else, by metamorphosis of the white blood-corpuscles into new connective tissue, to thickening, shortening and adhesion of the valves and tendon-threads (insufficiency and stenosis, valvular defects, chronic endocarditis). Sometimes, by detachment of the adherent growths of thrombi, it causes benign stoppages in the kidneys, brain, etc., which mostly scar over. The ulcerous form, on the contrary, ends in a complete necrosis of the tissue, which is more or less diphtheritically infiltrated.

Concerning the distribution of endocarditis upon the two halves of the heart, the proportion is obviously different in man and domestic animals. Whereas in man it usually attacks the left heart, in animals the right side is by no means rarely affected ; indeed it would almost seem to suffer more frequently than the other. Thus, according to Meyer, in nine cases of endocarditis the tricuspid valves were eight times implicated, and the bicuspid only four, the valves of the aorta being affected in two cases. Particularly among cattle do we find endocarditis of the right side, whereas among men this is extremely rare and mostly of fœtal origin. According to Jensen, it is generally the aortic or the bicuspid valves which are affected in the dog, the tricuspid and pulmonary valves more rarely.

Symptoms.—Acute endocarditis reveals itself first in tempestuous action of the heart, with palpitating, irregular and, later, diffused beat. The number of heart-beats sometimes exceeds that of the pulse, so that we find, for instance, in horses the proportion will be as 160 to 70. The action of the heart may be so vehement that the whole body is shaken with every stroke. The pulse is also rapid, and its speed may be between 80 and 160 a minute in horses and cattle, and in dogs may vary from 120 to 240. It is irregular, frequently intermittent and very weak, sometimes imperceptible. The heart-sounds are at first normal, but soon become abnormal, the first one in particular being very dull and sometimes amalgamating with the second to form one sound only. Later, certain characteristic endocardiac after sounds often arise, above all a blowing, contracted (systolic) noise with the first heart-sound, and more rarely—though of greater pathognostic importance—certain prolonged (diastolic) rustling and vibrating sounds. In acute endocarditis the temperature is always raised to fever point, and is especially high in the septic form. Trasbôt found in horses a temperature of 104° and 106° F.

Breathing is often so extremely laboured and accelerated that we might at first mistake the disease for pneumonia or pleuritis, and in some cases the respiration rises to decided dyspnœa. The general condition of the sick animal is very bad ; with serious brain affection, we observe great lassitude, weakness, and periodical fits of colic. To these come symptoms of metastasis in the lungs (attacks of choking), in the kidneys (bloody urine), in the extremities (lameness) and in the brain (apoplectic fits). In the septic form a general hæmorrhagic diathesis is developed.

Course.—Sometimes this is very acute, ending in death in a few hours or days ; at other times the malady endures for weeks, and an acute endocarditis may eventually develop into the chronic type. In the latter case acute inflammatory attacks readily recur. The result is always different according to whether we have a benign, verrucose, or severe, septic, ulcerous endocarditis to deal with. In the latter a fatal ending through development of a general septic infection, or of metastatic foci in the lungs, kidneys or brain, is much more frequent than with the verrucose form, which, nevertheless, leaves behind it serious changes in the shape of valvular defects.

Differential Diagnosis.—Acute endocarditis may, when its course is very acute, be confounded with anthrax, and also with septicæmia, inflammation of the brain, petechial fever, etc. On the other hand, owing to the extreme difficulty of breathing, it is not easy to distinguish from incipient pneumonia, from congestion of the lungs and œdema of the lungs. More difficult, however, is a differential diagnosis between endocarditis and myocarditis. In all these cases we must rely on thorough examination of the heart. Without the after-sounds, a diagnosis of endocarditis is impossible. We must remember that systolic after-sounds especially occur also in anæmia and leucæmia (so-called inorganic murmurs), whereby the diagnosis of endocarditis is rendered still more uncertain.

Therapeutics.—The treatment of acute endocarditis consists in avoidance of all excitement or movement, regulation of the action of the heart by digitalis, reduction of the fever by salicylate of soda, antifebrin, antipyrin and other fever remedies. In small animals an ice-bag may be laid upon the region of the heart, and in larger ones the same part may be permanently irrigated with cold water. If strength fail rapidly, use camphor, ether, spirits of wine, atropine, hyoscyamine, caffeïn, etc.

CHRONIC ENDOCARDITIS. VALVULAR DEFECTS.

Etiology.—Valvular defects are extraordinarily common among our domestic animals, especially dogs, horses and pigs. In by far the larger number of cases they are traceable to organic changes in the valves produced by chronic endocarditis, and chronic endocarditis of this description usually develops from the acute condition. Thus, for instance, many valvular defects in cattle spring from a previous general septic infection with acute articular rheumatism and simultaneous acute endocarditis. In horses such defects have been observed after acute infectious disease, such as influenza; and in pigs as a complication and sequel of erysipelas. Apart from this acute development, there is also a creeping, chronic endocarditis, caused by repeated chill, over-exertion or excitement. In pigs chronic endocarditis sometimes occurs without any precedent erysipelas. Arterio-sclerotic processes at the root of the vessels of the heart will lead to chronic changes in the valves affected,

particularly in old age. Hereditary pre-disposition of antenatal developments must be taken as the origin of many valvular defects.

Congenital Defects.—New Growths.—A minority of defects in the valves do not arise from endocardiac processes, but from congenital faults or new growths in the heart. Thus such inborn defects occur in the septum of both ventricles, and in the cow the septum may be absent entirely. Behrens noticed a perforation of the mitral valves in a horse, which was accompanied by a systolic after-sound and led eventually to hypertrophy and dilatation of the heart. In a dog which died suddenly, Jungers found a polypous new growth with long stalk, which firmly closed the left auriculo-ventricular orifice. Hink discovered in a cow, as the cause of sudden death, a fibro-sarcoma which had been detached from the wall of the left ventricle and completely stopped up the aorta. Franck saw an imperfect stoppage of the foramen ovale in a dog which suffered from epileptiform fits and general cyanosis. New growths in the heart or its vicinity, such as sarcomata, carcinomata, fibroids of the valves, myxomata or ulcers in the bronchial glands, may lead to stenosis of the valves of the heart. As the last-named conditions are of rare occurrence, we shall here treat only of such valvular defects as are caused by chronic endocarditis.

Post-Mortem Conditions.—Changes in the valves produce either inability to shut (insufficiency) or narrowing (stenosis).

1. The *insufficiency* of a valve arises from shrinkage, as well as shortening of its tendinous fibres. In the course of chronic endocarditis a change in the connective tissue of the valves occurs, with shrinking and atrophy, even complete disappearance. Owing to the simultaneous shortening of their tendinous fibres, the valves cannot distend to their full size, the orifice is thus enlarged, and even when the valve is closed, an open cleft is left, through which the blood is driven back again to the other side of the valve. Perfect or independent insufficiency or stenosis is rare, as the endocardiac process mostly leads both to inability to shut and to contraction. We therefore generally find stenosis and insufficiency side by side (combined valvular defects). Secondary anatomical changes in the body produced by valvular defects in consequence of disturbed circulation are: hypertrophy and dilatation of the heart; engorgement of the liver, brown induration of the lungs, engorgement of the kidneys and of the spleen; dropsies, especially ascites, hydro-thorax, hydropericardium, anasarca; emaciation; and diffuse hæmorrhage.

2. *Contraction of the Orifices* is produced by the thickening of the valves and also by proliferation of the endocardium.

The lumen of the orifice is found to be narrowed by polypous, wart cauliflower-like growths from the valves, or almost filled by tumour-like masses. The edges of the valves are thickened and spongy, sometimes covered with chalky incrustation, and the several valves grown together at their free edges. They have thereby lost their free mobility as well as their power of full distension, and are changed into rigid, unyielding, narrow rings, which at times are as hard as cartilage or bone. Consequently the normal quantity of blood cannot pass through them in a certain time and must therefore be forcibly driven through.

Occurrence.—These valvular defects are common among our domestic animals, occurring most frequently in aged dogs and horses, also in pigs. A large number of draught-horses and draught-dogs are incapacitated by them. The abundant literature on valvular defects in cattle is a result of the frequency of acute articular rheumatism among them: such defects have also been noted in birds. The distribution of the lesions between the two sides of the heart in domestic animals is about equal. Among 37 cases of endocarditis described in veterinary literature, 20 were in the left and 17 in the right heart. These were further sub-divided: 14 affected the mitral, 6 the valves of the aorta, 15 the tricuspid and 2 the valves of the pulmonary-arteries. The tricuspid valves seem therefore to have suffered a little oftener than those of the mitral, a proportion which holds good, especially in cattle. Among 7 cases in cattle, 6 were on the right side, and 1 was on both; none on the left heart alone. In horses the proportions seem to be about the same as in man: of 15 cases, 7 were on the left side, 5 on both sides, and 3 on the right.

That valvular defects are of extremely frequent occurrence, not only in dogs, but also in horses, is proved by a summary prepared by Nocard. In 42 old horses bought for purposes of operation, he found localised changes at the root of the aorta in 38, and in four others chronic endocarditis on the crescent-shaped valves of the aorta, and also upon the mitral valves. The intensity of the changes varied in individuals, the diseased valves being in some merely thick and stiff, in others drawn as by a cicatrix, and covered with fibrous vegetation, and again they were corroded, denticulated and perforated in many ways. The changes had been diagnosed during life. In all the animals there was a hissing sound, which began with diastolic murmur, and ceased with the first heart-sound. This sound varied in intensity and timbre. Cadiot and Ries found among 14 cases of valve-defects in horses that the aortic valves were affected in 10, the mitral-valve in 1, and that both were affected in the other 3. Among 13 cases in dogs, 5 were on the mitral, 4 on mitral and tricuspid, 2 on the latter alone, 1 on the aorta and 1 on aorta and mitral. Malkmus

and Bambauer describe a case of primary insufficiency of the tricuspid in a horse, without previous endocarditis, which was probably due to dilatation and hypertrophy of the right heart.

General Symptoms of Valvular Defects.—Both stenosis and valvular insufficiency cause an engorgement of the blood-stream. Defects in the mitral occasion engorgement in the pulmonary veins and lungs; in the tricuspid the engorgement is in the systemic veins. If the aortic or pulmonary valves be affected there will be an engorgement of the corresponding ventricle. These disturbances of circulation are at first equalised by increased activity of the heart, *i.e.*, of the divisions concerned, so-called *compensatory hypertrophy of the heart.* A defect of the heart thus compensated may exist for years without any serious morbid symptoms appearing, and only after severe exertion or excitement can any marked disturbances of circulation be perceived. In process of time loss of strength and weariness of the muscles of the heart show themselves; whereupon pronounced indications of engorgement of the lungs, and also of the vena cava, appear. In this stage only do the patients come under observation and treatment; the aspect of the malady is the same then as in the later stage of hypertrophy of the heart. The symptoms of a decreasing heart-compensation are:

1. *Acceleration of pulse and heart-beat,* easily excited activity of the heart, irregularity of pulse and heart-beat, palpitation. In isolated cases we find the contrary with the pulse, viz., diminished frequency (Bradycardie).

2. *Difficulty of breathing,* and after much movement dyspnœic attacks. This shortness of breath, which becomes a prominent symptom of the complaint, may be explained by engorgement and slower circulation of blood in the lungs, with subsequent permanent changes in that organ (brown induration of the lungs, contraction of the lumen of the pulmonary alveoli by the dilated lung capillaries).

3. *Increasing weakness, general emaciation,* slight sweating after brief exertion, but no fever. With draught animals, conspicuously rapid exhaustion during work is one of the earliest signs of incipient weakness of heart.

4. *Attacks of dizziness,* particularly after severe exertion (frequent in draught-dogs).

5. *Cyanosis* of the visible mucous membranes, in pigs also of the entire skin, particularly on the ears and back; great distension of the visible veins (those on the jaws, jugular veins); venous pulse.

6. *Hydrops of the bodily cavities* (ascites, hydrothorax, hydropericardium), as well as of the subcutaneous connective tissue (œdema on belly, evacuatory organs, chest and legs).

7. *Albuminuria and diminished secretion of urine,* being a sign of engorgement of blood in the kidneys.

8. Sometimes a *jaundiced discoloration* following upon engorgement fever.

9. *Emboli in peripheral organs,* arising from emboli in the heart, also in the lungs, brain, kidneys, liver, spleen and extremities (lameness, swelling of the joints).

10. Of pathognostic importance for the symptomatology of valvular defects are, finally, the *abnormal sounds, perceived on auscultation of the heart,* which will be more fully described later.

Symptoms of the Several Valvular Defects.—The most important of such defects are the stenosis and insufficiency of the mitral valves (horses and dogs) and tricuspid valves (cattle), also of the aortic valves (horses); much less common are defects in the pulmonary.

I.—INSUFFICIENCY OF THE MITRAL VALVE.

This, commonest of all valvular defects in horses and dogs, causes a back-flow of blood during the systole through the unclosed orifice into the left auricle, whereby the whirling motion of the retreating blood, as well as its contact with the venous blood, produces a louder or softer blowing, hissing or whirring after-sound, which is sharply systolic. The left auricle being fed from two sides, and therefore very full, becomes dilatated and hypertrophic. The discharge of blood from the lung-veins is hindered, and an engorgement takes place in these veins, in the lung-capillaries, and even in the lung-arteries, and consequently also in the right auricle and right ventricle, bringing in its train hypertrophy of the heart. As a result of the high pressure existing in the pulmonary artery, the second heart-tone (pulmonary tone) grows louder, more flapping, as it is usually expressed, and accentuated. Should compensation on the part of the right heart decrease, then cyanosis, dyspnœa, congestion of the jugulars, venous pulse, hydrops, etc., etc., set in.

Characteristic symptoms of mitral insufficiency are: systolic after-sounds, abnormally strong diastolic pulmonary tone, hypertrophy of the right side of the heart, feeble pulse, shortness of breath, engorgement of the venous system.

II.—STENOSIS OF THE ORIFICE OF THE MITRAL VALVES.

In contraction of the left auriculo-ventricular opening during diastole blood cannot pass unchecked from the auricle into the ventricle, but is only partially forced through the narrow opening with a whirling motion. Thus a diastolic (pre-systolic) after-tone is produced, the left ventricle is only partly filled and shrunken, and the pulse very small and feeble. The left auricle is dilated and hypertrophied, engorgement sets in throughout the pulmonary system, leading to hypertrophy and dilatation of the right heart; in consequence of which the pulmonary tone is much accentuated.

Characteristic symptoms of mitral stenosis are: diastolic after-sound, loud pulmonary tone, very small pulse, grave signs of engorgement, especially on the part of the lungs.

III.—INSUFFICIENCY OF THE TRICUSPID.

Inability of the right auriculo-ventricular valve to close causes a regurgitation of blood from the right ventricle into the right auricle, combined with a systolic after-sound. The backward movement of the blood in the auricle is carried on into the veins and causes a venous pulse, with engorgement in the venous system (cyanosis of the visible mucous membranes). The right auricle becomes dilatated and hypertrophied, and later the whole heart is distended. On relaxation of the latter, engorgement of blood in the lungs takes place, with formation of thrombi therein (dyspnœa), as well as in the venæ cavæ (hydrops).

Characteristic symptoms of tricuspid insufficiency are: systolic after-sound, venous pulse.

IV.—STENOSIS OF THE ORIFICE OF THE TRICUSPID,

which is rare in man, though frequent in cattle, is recognised by a diastolic after-sound, great repletion, dilatation and hypertrophy of the right auricle, venous pulse, retardation of the blood-stream in the pulmonary artery with subsequent formation of thrombi, and embolic conveyance of the thrombi into the lungs; hypertrophy, and also enlargement of the right ventricle, disturbance of breathing, and symptoms of dropsy.

V.—INSUFFICIENCY OF THE AORTIC VALVES

is rather common in horses, and causes a back-flow of blood during the diastole from the aorta into the left ventricle. Since a part of the blood therefore is driven back and meets that which issues from the left auricle, a diastolic secondary or after-sound is produced, which may also be felt as a whirring sensation. The overcharged condition of the left ventricle produces its extreme dilatation and hypertrophy. The hypertrophic state of the left heart causes also a strong arterial pulse, the curve of which, however, sinks abruptly owing to the retreat of a portion of the blood into the left ventricle: pulsus celer, *i.e.*, a rapid, jumping pulse. The diastolic aorta-sound is sometimes audible even in the carotid.

Characteristic symptoms of insufficiency of the aorta are: buzzing, diastolic after-sounds, extreme dilatation and hypertrophy of the left heart, strong arterial pulse, pulsus celer and arterial sounds.

VI.—STENOSIS OF THE ORIFICE OF THE AORTIC VALVES

sets the blood which streams through the narrowed valves into whirling movement, producing thus a systolic after-sound, when the pulse becomes slower and weaker, and the left ventricle hypertrophic. In extreme stenosis, symptoms of anæmia of the brain, vertigo, etc., appear.

Characteristic signs of aorta-stenosis are therefore: systolic after-sound, slow and very small pulse, hypertrophy of the left side of the heart, cerebral anæmia, etc.

VII.—INSUFFICIENCY OF THE PULMONARY VALVES

is very rare, and gives rise to a diastolic after-sound, with hypertrophy and dilatation of the right ventricle, and, on decrease of the activity of the heart, to extreme disturbance of respiration and circulation.

VIII.—STENOSIS OF THE ORIFICE OF THE PULMONARY VALVES

produces a systolic after-sound, hypertrophy of the right side of the heart, engorgement in the venous system, followed by difficulty of breathing, owing to disturbed compensation of the heart.

Diagnosis of Valvular Defects.—The recognition of a valvular defect is difficult for several reasons. First the after-sounds, both systolic and diastolic and without any organic change in the valves, occur also in leucæmia and anæmia as so-called un-organic heart-tones. On the other hand, these after-sounds may be lacking, especially at the beginning, even where there is a genuine valvular fault, and thus the most important diagnostic factor be missing. Two or more valvular defects may exist side by side in every possible combination, whereby the symptoms of the one may be masked by those of the other. Thus, stenosis and insufficiency are most frequently found together. In the last stage, viz., that of incipient disturbance of compensation, the symptoms of all are so very much alike (disturbed circulation in the lung and venous systems) that differential diagnosis is rendered extremely difficult, and it is unfortunate that in this stage only is attention first called to the ailment. Frequently one valvular defect so far predominates over the rest in its symptoms as to enable it to be at least approximately diagnosed. In order therefore to furnish a few points on which to base a differential diagnosis, the following should be noted as marking the several defects :

1. A *systolic after-sound* is detected in mitral insufficiency, tricuspid insufficiency, aortic stenosis and pulmonary stenosis. The first valvular defects are somewhat frequent, the last one is scarcely ever observed. Insufficiency of the tricuspid occurs most frequently in cattle and is associated with a venous pulse. Mitral insufficiency is found more in horses and dogs, accompanied by a weak pulse ; stenosis of the aorta with a very small and at times retarded pulse.

2. *A diastolic* (*pre-systolic*) *sound* may arise from stenosis of the mitral or tricuspid, and from insufficiency of the aorta or pulmonary. The latter is so rare as to be hardly worth consideration. Tricuspid stenosis is most common in cattle, with a venous pulse and severe dyspnœa, caused by formation of thrombi in the right heart. Mitral stenosis may be distinguished from aortic insufficiency by the small and irregular pulse, which in the latter is very strong and tends to become rapid. The diastolic sounds in horses and dogs is most frequently traceable to insufficiency of the aorta.

Therapeutics of Valvular Defects.—A compensated valvular defect requires no treatment ; we must merely feed the animals well, and guard them against over-exertion

and excitement. Not until the hypertrophied heart begins to relax in its activity, and disturbances of circulation are thereby set up in the lungs and venous system, must recourse be had to such remedies as may regulate the heart, reduce the excessive speed of the pulse, and increase the blood-pressure in the arterial system, thus giving the heart the pauses necessary for recuperation and reception of nutriment. For such purposes digitalis is a valuable drug, as it equalises the disturbed circulation, raises the arterial blood pressure and lessens the frequency of the pulse. It is given either as powder, or as an infusion of one-third strength, several days in succession. Its operation must, however, be carefully watched, and its use then discontinued for a while. The doses must be small. For example, we give a dog an infusion of from 15 to 30 grains fol. digitalis in eight ounces of water, daily one to two tablespoonfuls (for small dogs say teaspoonfuls); horses, 30 to 75 grains; cattle, 1 to 2 drams pulv. fol. digitalis upon bread, or in saline remedies. Besides digitalis, caffeïn is to be recommended when much dropsy is present, it having the advantage of not being poisonous when administered for a considerable length of time, and possessing a stronger diuretic effect. Give it in the fo m of its salts, *e.g.*, caffeïne sodio-benzoate, to dogs daily 4 to 15 grains, horses and cattle 1 to 2½ drams. Tincture of strophanthus acts similarly (horses and cattle 2½ to 6 drams, dogs 10 to 25 drops); also diuretin (dogs every two hours 4 to 8 grains); and theocin (3½–15½ grains for dogs). Iodide of potassium is highly recommended as an adjuvant to digitalis (horses 1 to 2½ drams).

From a symptomatic point of view, puncture is advisable where there is excessive dropsy in the cavities (ascites, hydrothorax, hydropericardium); also a diuretic treatment (the above-named heart-specifics, diuretin, scilla, acetate of potash), and a diaphoretic or saliva-stimulating course (pilocarpine, arecoline). Drastics may also procure temporary relief. To combat general weakness, stimulants should be used, and against abnormal palpitation, narcotics, especially morphia in subcutaneous injection (horse 8 grains, dog ⅛ to ⅜ grain).

MYOCARDITIS.—INFLAMMATION OF THE MUSCLES OF THE HEART.

Etiology.—The causes of inflammation of the muscular structure of the heart are to be sought, firstly, in over-exertion (long-distance rides and drives), the excitement which follows parturition in horses (especially blood-horses); and, secondly,

and more rarely, in chill. Myocarditis is, however, usually a secondary condition resulting from general infective disorders, such as anthrax, foot-and-mouth disease, septicæmia, pyæmia, tuberculosis, etc. An inflammation of the endocardium or pericardium may extend to the myocardium. Injury to the heart, which is very common in cattle, though always complicated with pericarditis (see earlier in this chapter), may also produce myocarditis, as in horses, and may result from shock or contusion of the muscles of the heart.

Post-Mortem Conditions.—The changes in the myocardium are extraordinarily varied, according as a parenchymatous, an interstitial, an indurative or an abscess-forming myocarditis be present. Whereas in interstitial and abscess-forming myocarditis the inflammation is usually focal, circumscribed and even disseminated, in the parenchymatous form it generally occurs diffusely.

1. *Acute parenchymatous myocarditis* is rarely found as an independent and primary disease. The inflammation attacks not only the muscular fibres, but is complicated with interstitial infiltration. Viewed in the mass, the muscles show a spotty discoloration, and appear from greyish-red to greyish-yellow, chalky and even whitish-yellow, of tender consistence, and their fibres seem obliterated, indistinct and homogeneous. The microscope reveals a loss of the correct striation, as well as a dusky opacity in the muscular fibres, with considerable small-cellular infiltration in the interstitial connective tissue, the muscles being thrust apart by a layer of white blood-corpuscles.

2. *Chronic indurated myocarditis* appears in the form of whitish, shining, so-called callosities of the heart, which are like connective tissue in texture, and sometimes as hard as cartilage, also of tendinous deposits (milky and tendinous spots, macula lactea). These vary in size and penetrate the muscles of the heart, especially the left near the apex, being found sunk beneath its sectional surface, and occasionally making its walls so thin that they cause the exterior to bulge (aneurysm of the heart). We also find the fibrils of the muscles sometimes fattily-degenerated, granular and surrounded by an abundant growth of connective tissue. The whole develops from a small-cellular infiltration of the interstitial connective tissue, with induration and cicatrisation, and leads usually to secondary hypertrophy of the heart.

3. *Purulent myocarditis, or abscess of the heart,* occurs chiefly in pyæmia :—in cattle with puerperal metritis, in foals with omphalo-phlebitis. The abscesses are either miliary and disseminated through the cellular connective tissue ; or larger, varying from a hazel to a chestnut in size, appearing in conjunction with an embolic stoppage (metastatic myocarditis). Abscesses of the heart have been observed in both cattle and horses ; in the latter they sometimes become caseous and even show a chalky coating (ossification of the heart).

Degeneration of the Muscles of the Heart.—Closely related to myocarditis, and to some extent resembling its first stage, although much commoner, is the degeneration of the muscles of the heart occurring in the course of infectious diseases :—opaque swelling, fatty degeneration, etc., whereby the heart as a whole appears very tender and flabby, as though boiled, and is also discoloured. On the other hand, there is no small-cellular infiltration. The clinical aspect of the complaint is similar to that of myocarditis, and both of them produce insufficiency of the muscles of the heart. The so-called callosities of the heart are not always developed upon an inflammatory base, but arise, according to Ziegler, much more frequently from a myomalacia, *i.e.*, from softening of the myocardium, following on anæmic necrosis, a condition which has hitherto been erroneously termed parenchymatous myocarditis. This myomalacia cordis develops from a hæmorrhagic stoppage caused by arterio-sclerotic or embolic processes, with which are complicated, one after the other, arterial anæmia, softening, caseation, resorption of the waste matter with final cicatrisation. In a similar way scabs of connective tissue form on circumscribed foci of softening in the brain, or in stoppages of the kidneys.

Symptoms.—Are those of disturbance of function of the cardiac muscles, *i.e.*, of insufficiency or weakness of the heart, and correspond with the symptoms of dilatation of that organ. The heart-beat is at first very tumultuous, throbbing, extended, and at times is both visible and audible at some distance ; it is also irregular ; later it grows very feeble. The pulse is very rapid, small, irregular and often imperceptible, counting sometimes in horses continuously from 80 to 120 strokes a minute. Respiration grows difficult, rapid, and is succeeded by extreme weakness and prostration, trembling, fits of dizziness and great excitability. Temperature is raised in accordance with the generating cause. In cases of long duration, general appearances of engorgement set in, as with hypertrophy of the heart. In other instances the inflammation of the myocardium takes a very acute course, and the animals suddenly and apoplectically collapse, many cases of foot-and-mouth disease

or of tuberculosis ending thus from paralysis or rupture of the heart. Both acute parenchymatous and suppurative myocarditis commonly end in this way.

Differential Diagnosis.—The differentiation of primary myocarditis from other heart-affections, and especially from passive enlargement, endocarditis and pericarditis, is very difficult, indeed often impossible, as the symptoms are almost identical. For these reasons a diagnosis is especially difficult whenever the characteristic signs of pericarditis, endocarditis or a valvular defect are not present. In general the probabilities would favour a diagnosis of myocarditis when severe phenomena of heart-insufficiency are noted without any demonstrable organic change (after-sounds, rubbing noises). The identification of a secondary myocarditis, such as occurs in contagious pleuro-pneumonia, is far eaiser.

Therapeutics.—The treatment for inflammation of the heart corresponds with that for weakness of the same organ, which is in avoidance of all excitement or exertion, strengthening the muscles by nourishing food, stimulants, digitalis, caffeïn, strophanthus, camphor, atropine, hyoscyamine and alcohol, and administration of febrifuges. Zschokke gives strong wine (2–4 bottles a day) to horses with weak hearts and, in feverish conditions, iodide of potassium (2½–3 drs. once a day). In cases with very acute course treatment is of no avail.

New Growth in the Muscles of the Heart.—Among these, sarcomata seem to be of special clinical importance. In a dog which died with symptoms of weak heart, dissection revealed grey, circumscribed foci in the myocardium, which were found under the microscope to be a sarcomatous infiltration of the fleshy part of the heart, with atrophy of the muscular fibres. At the same time there were multiple, metastatic, sarcomatous foci in the kidneys (Fröhner). Sub-epicardial fibro-sarcoma are by no means rare in slaughtered oxen (Ostertag, Kitt, Fehsenmeier). In an army horse which died suddenly while trotting, a lipoma weighing 3½ lbs. was found in the heart. In a horse and an ox which both died suddenly, a carcinoma and a myxoma, respectively, were found in the heart (Larrue, Bissauge). In a cow, which had shown extreme dyspnœa, a fibroma, which had become detached from the mitral valve, was found wedged in the aortic arch (Hemminger). Thompson observed the same thing in a mare (frequent falling down and cramp). An osteosarcoma, as big as a man's head, was discovered by Doenecke in the wall of the right auricle.

HYPERTROPHY AND DILATATION OF THE HEART.

General Observations.—Hypertrophy and dilatation of the heart in their advanced stages are anatomically and clinically distinct, and should be discussed in different chapters. In practice they can only be differentiated with difficulty, because they very often occur together, viz., with valvular defects; or, after having for a short time existed independently, speedily pass one into the other; also because in the later stages of disease a differential diagnosis between them is usually impossible, owing to the similarity of their symptoms. The relationship of the two is further expressed in our terminology, which describes hypertrophy of the heart as active, and dilatation as *passive enlargement of the heart.*

Post-Mortem Conditions.—Both hypertrophy and dilatation cause an enlargement of the heart; in the former this increase of volume takes the form of a growth in the substance of the cardiac walls, and the latter in enlargement of its hollow cavity.

1. The enlargement of the heart in *hypertrophy* can only be produced by a thickening of its walls; which somewhat rare form we term a simple hypertrophy of the heart. In by far the greater number of cases this increase of thickness is accompanied by an enlargement of the cavity, so-called eccentric hypertrophy or active enlargement of the heart, which is usually what is meant when we speak of hypertrophy of the heart. Whether a third form exists in which the cavity is diminished while the walls are thickened, or a so-called concentric hypertrophy of the heart, is doubtful. By the term of false or spurious hypertrophy we understand an enlargement caused by foreign deposits within the cardiac walls. True hypertrophy of the heart may be either total or partial, viz., affecting one ventricle only, or it may be circumscribed and confined solely to one papillary muscle. The two latter forms are most common. The left heart is by far most frequently hypertrophied. The shape of the heart then becomes elongated and cylindrical, whereas in right-hand hypertrophy it is more flattened and broadened, while a totally hypertrophied heart is almost round and often attains colossal dimensions. Thus Stephenson found a horse's heart weighing 32 pounds, and Gerlach one of 19 pounds; Herran measured the heart of an

ox which was 14½ inches long and weighed 36 pounds, the walls being incrusted with lime-salts. The wall of the left heart is not unfrequently more than twice or thrice its usual thickness. Its structure may nevertheless remain quite normal, although hypertrophied. As a rule, the muscles are harder, of tougher, firmer consistency, sometimes penetrated by proliferations of interstitial connective tissue, being also darker (brownish-red in colour), and may later be marked with lighter spots and stripes (deposits of pigment; fatty degeneration of the muscular fibres). The wall of the right heart when hypertrophied gapes if cut open, instead of collapsing.

2. *Simple dilatation of the heart* produces enlargement essentially by distension of its cavities, by which its walls must naturally become weaker and thinner. Relaxed and distended walls are characteristic of passive enlargement of the heart, and whereas in the active form the organ gains in strength, in the passive it loses it. Dilatation of heart is usually found on the right side. In dilatation of the left side the walls collapse on being cut, instead of gaping. The structure of the myocardium may be normal, but the wall is usually relaxed, anæmic, fattily-degenerated, friable and easily torn, of pale, yellowish-brown colour and very thin, indeed in some places quite transparent, consisting only of endocardium and epicardium. With these appearances a bulging-out of the wall may also be developed, so-called aneurysm of the heart. In extreme cases of dilatation the heart may be more than double its normal size.

Etiology.—The causes of eccentric hypertrophy of the heart are usually to be sought in circumstances which offer resistance to the circulation and thus increase the blood-pressure. Among them we must mention :—

1. *Increased physical exertion*, so-called *labour hypertrophy or idiopathic hypertrophy* of the heart, which occurs particularly in race-horses as a result of very severe—and though brief, yet often repeated—exertion; also in draught oxen and fox-hounds. This is the reason why a race-horse's heart is larger than that of under-bred horses. Thus, for instance, the heart of the English thorough-bred stallion Helenus weighed 13¼ pounds. The development of idiopathic hypertrophy of the heart has been explained by assuming that the contraction of the muscles of the trunk during hard work leads to contraction also of the muscular arteries, and thus to an increased pressure

of blood, which necessitates greater activity on the part of the heart, causing a muscular hypertrophy. A physiological cardiac hypertrophy occurs during, and sometimes after, pregnancy (transient and permanent hypertrophy of gestation in cattle).

2. *Valvular defects* of the heart lead frequently to hypertrophy of that organ, owing to the disturbance they cause in the circulation; this is a so-called *compensatory* hypertrophy, because such disturbances are supposed to be balanced by it.

3. The various *obstacles to circulation in the blood-vessels* cause secondarily a so-called *symptomatic* hypertrophy of the heart. Under this head belong aortic aneurysms, congenital stenosis of the aorta, thrombi, compression of the aorta by new growths, atheromatous and arterio-sclerotic processes, excess of blood (plethora).

4. *Lung-diseases*, such as emphysema of the lungs, compression by exudations or transudations, adhesion to the costal walls, interstitial glanderous or tuberculous pneumonic processes, which shut off part of the pulmonary vessels from circulation, increase the blood-pressure in the region of the right heart, and thus give rise to a hypertrophied heart.

5. *Adhesion of the heart to the pericardium* also increases the demands upon the activity of the heart by impeding its free action, and thus causes it to grow in size.

6. Finally, *chronic inflammation of the kidneys* is almost always complicated with hypertrophy of the heart.

Passive enlargement of the heart, or *dilatation*, assumes usually the continuing stage of the active form, in so far that the hypertrophic heart is finally unable to meet the demands made upon it, its action grows weaker, and it becomes distended by the abnormally increased blood-pressure. In this way fatty-degeneration of the heart, myocarditis, endocarditis, sclerosis of the coronary arteries, changes wrought in the myocardium by high fever, infectious diseases, poison and anæmia all produce dilatation. A passive enlargement of the heart may develop acutely after acute lung-affections with serious interruption of circulation and extreme increase of blood-pressure in the region of the right heart. For instance, in acute pneumonia of horses, if long continued, an incipient hypertrophy of the heart may arise, which, however, disappears later. On the contrary, chronic changes in the lungs always relieve a hypertrophy of the heart, as has been already stated, unless the animals be too feeble; in the latter case, dilatation nevertheless occurs in spite of the chronic course, owing to the lack of sufficient nutritive material.

Symptoms.—A genuine, and especially a compensatory hypertrophy of the heart usually develops so gradually that it may exist for years without any visible disturbance of the general health. In extreme cases, as, for instance, during chronic nephritis, it reveals itself by a full pulse, exceedingly powerful heart-beat, very loud and clear heart-sounds and in extension of the cardiac dulness. This, the sole characteristic symptom of "hyperkinese," of hypertrophy of the heart, does not, however, make its appearance very often, because the hypertrophy sooner or later passes into dilatation and exhaustion, owing to gradually increasing insufficiency in the action of the heart, and because an idiopathic hypertrophy, which is not produced by other organic changes nor complicated therewith, is of rare occurrence. Hence the cases of illness designated by the name of "Hypertrophy of the Heart" in literature and in practice mostly refer to complications of active and passive enlargement of the organ, often to the latter alone. The morbid symptoms of this double affection, which are exactly those of non-compensated valvular defects, differ considerably from those of the true hypertrophy of the heart as named above.

The symptom which is common to both active and passive enlargement of the heart is extension of cardiac dulness as demonstrable by percussion. With great dilatation, *e.g.*, in dogs, this may reach to the region of the last false ribs. Incipient exhaustion of the hypertrophied heart then shows itself in oppressed breathing, palpitation, an abnormal pulse, hyperæmia and anæmia of the brain, fits of dizziness, as well as in extensive disturbances of circulation with their consequences. The most prominent sign is the strong throbbing of the heart, which can be felt on both sides, and is even visible from a distance shaking the whole body. The heart-sounds are irregular, the first very loud, with metallic ring and sometimes vibrating, and the second mostly weak or even quite absent. The pulse, in marked disunison with the heart, is generally weak or even quite imperceptible (hypokinesis); after very slight exertion extremely rapid, irregular and uneven. Sometimes a venous pulse can be observed in the jugular vein. Even when at rest, breathing is quickened, and rises to dyspnœa if the animal be used (so-called cardiac asthma, pursiness), or even to suffocation. The animal staggers, falls, and dies of apoplexy. The later symptoms occur sometimes in the form of fits of dizziness, trembling, twitching, sweating and dyspnœic, stertorous breath.

The interruptions which occur later on in circulation soon affect the brain (congestion and anæmia of the brain in the form of vertigo and fainting), also the bronchial membrane (bronchitis), the digestive tract (chronic intestinal catarrh), the liver (engorgement of the same), and the kidneys (engorgement with albuminuria and oliguria). Thrombosis of the arteries (paralysis of the hind-legs caused by thrombosis in the region of the posterior aorta) is also a not uncommon sequel to disturbed circulation. General cyanosis and dropsy finally develop (ascites, hydro-pericardium, hydro-thorax, anasarca) ; the lower parts of the breast, belly and extremities swell, the animal grows very thin and either perishes of marasmus or of embolism of the lungs, caused by coagulation of blood in the heart, paralysis of the heart, hæmorrhage on the brain and carbonic-acid poisoning.

The symptoms just described are commonest in old dogs, which are brought to be treated for great shortness of breath, or for frequent dizziness, also in horses when examined for asthma or " thick-wind " ; they may also occur in other animals. Thus Herran reports on a case of hypertrophied heart in an ox, which gradually wasted away with the appearances of chronic hard-breathing, and was obliged constantly to rest after every exertion owing to tumultuous heart-throbbings.

Differential Diagnosis.—Hypertrophy and dilatation of the heart are often mistaken for a primary lung affection, *e.g.*, congestion of the lungs or pneumonia. Owners are particularly liable to be thus misled by the great prominence of the symptom of difficult breathing. A careful examination of the lungs by auscultation and percussion, the gradual appearance and long persistence of the condition, as well as a positive demonstration of some heart-affection enable us, however, to arrive at a correct diagnosis. Active and passive enlargement of the heart differ, moreover, from primary palpitation by absence of extended cardiac dulness, as well as of the grave consequences of the latter. The palpitation also is not continuous but intermittent. More difficult is the differentiation from hydro-pericardium and myocarditis, especially the chronic form of the latter, which is accompanied by induration ; indeed it is often impossible, as the symptoms of chronic myocarditis are precisely similar to those of hypertrophy of the heart. On the other hand, valvular defects may be differentiated from hypertrophy

with ease, as they exhibit characteristic heart-sounds, without which they could not be diagnosed.

Therapeutics.—The chief aim in prescribing for hypertrophy and dilatation of the heart is to determine their causes and remove or combat them. We must, however, observe that any hindrance of a compensatory hypertrophy which serves to balance existing defects is objectionable, especially the use of digitalis. In this respect the nature of the pulse, especially its magnitude, is of first importance for therapeutics. We must seek to avoid all exertion or excitement; horses must not be ridden nor worked, and dogs must not hunt, whereby indeed the value of such animals is so much depreciated that they are hardly worth further treatment. Above all, they must have good, albuminous nourishment, that the muscular apparatus of the heart, being compelled to hypertrophy, may have material for formation of new muscle. Of drugs we may give iodide of potassium as a sedative against the hyperkinesis of the heart which arises in the course of its hypertrophy. It is given to horses in doses of 1½ to 3 drams, and to dogs of 3 to 15 grains.

If, however, the hypertrophy be already complicated with enlargement, and the pulse therefore grown feeble, the exhaustion and weakness of the heart must be combated by cardiac stimulants, as well as by strengthening remedies. Digitalis-leaves are given then in small and frequent doses (horses, ½ to 1½ drams; dogs, 1½ to 5 grains per diem in infusion). As these tend to reduce the speed of the heart, the pauses between the various phases of its activity are longer and its muscles gain time to recover and absorb nutriment. Naturally the continuous administration of digitalis requires great caution. Besides digitalis we may mention as admirable cardiacs, caffeïn (horses and cattle, 1½ to 3 drams; small dogs, 1½ to 8 grains; larger dogs, 8 to 30 grains), and strophanthus-tincture (horses and cattle, 2½ to 6 drams; dogs 10 to 25 drops). Among stimulating remedies, especially if disturbances of circulation appear, we may use wine, camphor, alcohol, ether and veratrine. Good, substantial food acts as a strength-giver. By proceeding in this way we may at least delay for a time the impending insufficiency of the cardiac muscles, even though a perfect cure of hypertrophy and dilatation occurs only in rare cases, viz., where the cause can be permanently removed, as in idiopathic hypertrophy.

RUPTURE OF THE HEART.

Etiology.—The causes which lead to rupture of the cardiac muscles are, apart from traumatic influences, to be sought in some morbid condition of its walls, which leads either directly, or after the operation of certain other factors, to a solution of continuity of the organ. Among morbid changes of the myocardium must be cited: softening, inflammation of the cardiac muscles, formation of abscesses, weakening of the wall by induration and aneurisms, fatty or senile degeneration of muscular fibres, endocardiac and pericardiac processes, atheromatous degeneration of the aortic walls at their origin, echinococci cysts and worms (spiroptera) in the wall of the heart; The exciting causes are sometimes physical shock, such as a fall, over-excitement during coition, or (in dogs) during operation for ascites, and at other times acute tympanites, as often occurs in cattle, etc.

Post-Mortem Conditions.—Besides the changes mentioned in the myocardium, we find a fissure in the wall of the heart, very often in that of a ventricle or auricle, and from one to two inches in length. Occasionally there are several fissures. Thus Ismert observed 12 ruptures of the myocardium of a thorough-bred horse, the muscle having undergone fatty degeneration. Sometimes either the aorta or pulmonary artery is ruptured at its base. Blood is found collected in varying quantity in the pericardium (hæmopericardium).

Symptoms.—Rupture of the heart betrays its occurrence either in sudden, apoplectic collapse and death, the animal often uttering a loud cry, or in staggering, dizziness, trembling, quickened breathing, falling, and convulsions, accompanied by indications of internal hæmorrhage. Death in the latter case follows after some hours. No treatment is possible.

PALPITATION OF THE HEART AND SO-CALLED ABDOMINAL PULSATION IN HORSES.

Definition.—Under this name of "Palpitation" we find a series of morbid appearances in horses and dogs described in literature, which, being by no means uniform, have met with

very different interpretation and explanation. They cannot all be identified with the neurosis of the heart which in man we call palpitation, but fall partly under this head. The greater number appear to be entirely distinct from primary heart-affection, but identical with nervous cramp or spasm of the diaphragm. We discuss them here all together in the chapter of heart-diseases, because they are usually designated palpitation of the heart, and because their pathogenesis is as yet too little known for them to be assigned to a special chapter; moreover, rhythmical, spasmodic pulsation in the region of the thoracic and abdominal cavities is common to them all. Subsequent affections seem to have been described as "Palpitation of the Heart," or "Abdominal Pulsation" in the abundant literature which we possess.

1. **True Nervous Palpitation of Heart,** caused by neurosis of the heart without any demonstrable anatomical changes. The frequent accentuation of the heart-beat which occurs during hypertrophy of the heart, especially with valvular defects, does not come under a correct definition of palpitation, nor the throbbing, laboured heart-beat which accompanies anæmia and leucæmia, caused by disturbed nutrition or atrophy of the heart. The causes of nervous palpitation are to be first sought in a general nervous temperament of certain animals; while among directly operative factors are great excitement, from excessive exertion, severe flogging, hanging by night on the halter, hanging on the reins too much in riding, fright, etc.

The *symptoms* of nervous palpitation are: throbbing heart-beat, accompanied by concussion of the costal wall, or even of the entire thorax, and which may sometimes be heard a few paces away; accelerated, small, irregular pulse, quickened respiration, restlessness, fear, trembling, sweating. Auscultation of the heart reveals very loud heart-sounds, which are frequently amalgamated into one. The accentuated heart-beat can also be perceived along the course of the aorta by laying the ear against the animal's back. During the attack the animal often passes fæces, and poses itself for urination. Venous pulse and even visible pulsation of the carotid arteries have also been noticed. In isolated cases, the ailment ended fatally in paralysis of the heart or cerebral apoplexy; but in most instances, recovery takes place after a shorter or longer duration of the attack, although the paroxysms may be

frequently repeated. Of considerable importance in differentiating palpitation from kindred affections is an early test as to the correspondence between the throbbing in the thorax and the several heart-beats; the two should exactly synchronise.

2. **Nervous Spasms of the Diaphragm.**—These produce symptoms, closely resembling those of palpitation of the heart, with which they are often confounded by observers, or described as "*abdominal pulsation.*" Contractions of the diaphragm may have many causes. Catarrhal and inflammatory conditions of stomach or bowels seem able to produce spasmodic cramp of the diaphragm, either by reflex action, or by transmitting inflammation to that membrane. Thus, marked symptoms of palpitation of the diaphragm were noticed by Brill in conjunction with vomiting; Berghuis, after overfeeding; Oreste, with gastric phenomena, bad quality of food, colic, etc.; Grosswendt, in bowel-inflammation; Leblanc, Wörz and others, immediately after drinking cold water; Boiteau, after eating fungoid hay, in which case they were to some extent an indication of poisoning; and Cartwright, after administration of an aloes-pill. Lucet observed marked symptoms of palpitation of the diaphragm in a pregnant cow after greedy feeding. It is also possible that nervous palpitation of the heart may set up morbid contraction of the diaphragm, and that the phrenic nerve, which runs across the base of the heart, may be irritated by abnormal activity of that organ (Haubner-Siedamgrotzky).

The phenomena of spasms of the diaphragm—not inappropriately called also "Chorea of the Diaphragm"—are most peculiar and striking. In the first place we note violent throbs, especially in the region of the left inferior ribs and flanks, which shake the whole body. These are sometimes accompanied by a subsequent short backward expiration (Boiteau, Hering), and by a dull tone audible some yards off (Simonin and others). These throbs, resembling electric shocks, may be most distinctly felt along the insertion of the diaphragm (Grosswendt), and seem at times to be restricted to certain spots upon the latter (Cartwright). In rare cases they are accompanied by a decided sob (*singultus*). They do not synchronise with the heart-beat, but count only from 10 to 15 strokes per minute, seldom more, and in most cases the heart-beat is not at all violent, but normal or even unusually weak. It is evident then that the abdominal pulsations thus described cannot arise from the heart as prime

factor. Only in the minority of cases were these abdominal pulsations synchronous with the heart-beat, though always stronger, while the throbs decrease in violence forward, near the region of the heart, when it is likely that an irritation of the phrenic nerve by the heart movements may occur. Besides these symptoms, a small, frequent pulse is noted, increased and jerky breathing, tremblings, restlessness, yawning, frequent stretching out of a hind-foot with indications of pain. The spasmodic attacks occasionally last but a few minutes, at others from 12 to 24 hours, or even from 2 to 4 days, seldom longer, though Leblanc observed a case lasting three weeks. *The prognosis* seems to be less favourable than in palpitation. Cartwright noted six fatal cases among 18. The greater frequency of left-hand pulsation (Cartwright records that of 16 cases, 12 were on the left side) may perhaps be owing to the position of the stomach, or to changes therein producing illness. In one case, Duschanek observed nervous palpitation of the heart complicated with spasm of the diaphragm.

Treatment of Palpitation of the Heart and Spasms of the Diaphragm.—This consists in the administration of anti-spasmodic remedies, such as morphia (subcutaneous injection of 5 to 8 grains for a horse); bromide of potash and chloral hydrate (6 to 12 drams). In nervous palpitation of the heart, cold irrigation of the cardiac region may be recommended, with irritant inunctions upon the skin, besides derivation to the intestinal canal by means of purging and even drastic remedies. The efficacy of bleeding, once so regularly adopted, is uncertain.

NOTE:—In assuming, as we do above, that the origin of so-called abdominal pulsation is to be sought exclusively in spasm of the diaphragm, we agree with the majority of observers, the most eminent of whom, such as Apperley and Castley, Beeson, Boiteau, Goubeau, Hering, and, in later years, Cagny, all maintain this opinion. Confusion arose only when cases of true nervous palpitation of the heart were classed with spasms of the diaphragm. The acceptance of this last-named malady explains easily and naturally the pulsations, sometimes so extremely violent, whereas a mere interruption in the circulation in the posterior aorta (Berghuis, Simonin, Anacker and others) cannot possibly produce this phenomenon, still less so an aneurysm of the aorta. A simple contraction of the abdominal muscles (Délafond, Cadiot) cannot be considered, as it produces straining only. To deduce these pulsations entirely from the artery of the liver and kidneys is certainly a very naïve explanation. The palpitation noticed in connection with thrombosis

of the iliac arteries and posterior aorta is entirely distinct from the malady before us, although they have often been confounded. From this data, we must regard the so-called abdominal pulsation of horses as being a neurosis of the diaphragm, closely related to singultus in man, usually occurring alone, but sometimes also simultaneously with nervous palpitation of the heart.

Bradycardia.—Under this name we understand, in contrast to palpitation, a noticeable retardation of the heart's action, that is, of the pulse. Thus in horses, the latter will sink to 14–20, and in dogs to 20 beats a minute. The causes of this phenomenon are diverse (valvular defects, dilatation of the heart, enlarged aorta, new growths near the centre of the vagus, great loss of blood, feeding with new-mown hay). Zimmermann observed symptomatic bradycardia (9 beats) in a horse with cardiac degeneration, suffering from gastro-enteritis. The pulse-rate in a horse rose from 12 to 30 after giving sodium bromide (Zschokke).

ANEURISMS.

Etiology.—The causes of aneurisms of the aorta, *i.e.*, of circumscribed enlargement of that vessel, are mostly to be sought in morbid changes of the walls by chronic endarteritis and mesarteritis, accompanied by fatty and calcareous degeneration. Such vascular troubles occur in horses most frequently in the mesenteric arteries as a result of the presence of strongyus armatus, known as worm-aneurisms. (See the par. on Embolic Colic in Chap. III.) Spiroptera sanguinolenta may also produce the same result, as also may traumatic, mechanical influences, such as violent muscular exertion, a blow or a fall, particularly if some arterial disease be already present. A diathesis seems sometimes to be also present for diffuse formation of aneurisms (*Raymond*).

Post-Mortem Conditions.—Aortic aneurisms are either sac-shaped, spindle-shaped or cylindrical. In true aneurisms the sac is bounded by the walls of the vessel, and in false ones by those of neighbouring organs, such as the bowels, liver etc. The size may be considerable, exceeding that of the human head; and at times they grow fast to the adjacent organs, *e.g.*, to the stomach, colon, cæcum or liver. Aneurisms are commonest upon the anterior mesenteric artery, also at the bifurcation of the aorta, on the abdominal artery, the thoracic aorta, at the root of the aorta, on the coronary and femoral arteries, on the internal maxillary, etc. In most cases

all three coats of the aorta share in the dilatation ; being thereby atheromatously and fattily degenerated, alternately thickened and atrophied, and showing lamellar deposits of lime. The contents consist of coagulæ of fibrin, which are frequently stratified, organised, or already purulently decayed. According to Röll, these may in domestic animals spontaneously heal by shrivelling and cicatrisation. Since the distended aorta naturally presses upon the nearest organs, it compromises the lungs, stomach, viscera, liver, kidneys, and even affects the vertebral column. Finally, it may rupture and empty its contents into the thoracic and abdominal cavities, even into the stomach (Vogel, Bleich) and rectum (Labat and Cadéac).

Symptoms.—In most cases aneurisms of the aorta either produce no visible signs of disease, or end suddenly by hæmorrhage without any warning symptoms having been noted. The consequences of aneurisms of the anterior mesenteric artery have been already described in the section on embolic colic. Death by internal hæmorrhage occurs especially after severe exertion, the animal tottering, displaying great difficulty of breathing, collapsing and speedily expiring. In other cases, peculiar paroxysmal phenomena are noted, without any immediate fatal ending. Thus Lustig observed staggering of the hind limbs in a horse after severe exertion, sudden collapse, extreme dyspnœa, as well as epileptiform spasms, during which the head and neck were bent backwards and the feet stretched out. In from 5 to 15 minutes the animal recovered. In another case of aneurism of the pulmonary artery in a horse, Lustig noticed fits of dizziness. Barrier records, in a dog with aneurism of the posterior aorta, growing emaciation with ravenous appetite, increasing weakness and extremely hard breathing after the 'east movement and finally paralysis of the hind parts (signs of weak heart). In a horse which had an aneurism of the thoracic aorta as large as a man's head, Schmidt noticed a humming tone over the vertebral column which synchronised with the action of the heart, inability to pick up food from the ground, combined with a stiff and constrained gait in the hind limbs. (For rupture of worm-aneurisms, *cp.* Chapter on "Embolic Colic.")

Therapeutics.—In man, the treatment consists in the application of galvano puncture, which is even extended to the thoracic aorta ; in injections of ergotin, as well as internal

administration of sugar of lead, or iodide of potassium. These last remedies may also be applied to animals. Aortic aneurisms are, as a rule, incurable.

RUPTURE OF THE LARGER VESSELS OF THE THORACIC AND ABDOMINAL CAVITIES.

Etiology.—Ruptures of the larger vessels in the cavities of the thorax or abdomen generally indicate some change in the walls of such vessels caused by chronic endarteritic processes (arterio-sclerotic, or atheromatous changes), or aneurism. The indirect sources of such rupture are partly physical exertion, such as the drawing of heavy loads, and partly a fall, the act of vomiting, the operation of veratrin medicinally administered, etc. Ulcerative processes in the locality of the artery may also destroy its walls. In rarer cases vascular rupture is produced by the presence of worms in the vessels; thus by strongylus armatus in horses, or spiroptera sanguinolenta in dogs. Finally, animals with hypertrophy of the heart are pre-disposed to rupture their blood-vessels. In many cases, however, no demonstrable cause is to be found. This refers particularly to changes in the vessel walls.

Anatomical Notes.—Rupture of the larger vessels occurs in the aorta and its branches, in the portal vein, in the posterior vena cava, in the anterior vena cava, and in the pulmonary artery. Most aorta-ruptures affect the root of the vessel near its crescent-shaped valve, when the blood gushes into the pericardium, which, on dissection, is found filled with it. If only the intima and media be ruptured, the blood flows in between the media and adventitia, forming a so-called dissecting aneurism. Rupture of the intestinal vessels leads to the formation of hæmatoma between the folds of the mesentery.

Symptoms.—The signs of rupture of a larger vessel are those of internal hæmorrhage, shown by sudden paleness of all visible mucous membranes, thready or imperceptible pulse weakness, staggering, collapse, with cold extremities. In one case Straub saw epileptiform fits precede the rupture of an anterior vena cava. Hartmann found violent hæmoptysis in the bursting through of an aneurism of the pulmonary artery into a larger bronchus. Treatment of such internal vascular ruptures is impossible.

THROMBOSIS OF THE FEMORAL, PELVIC AND AXILLARY ARTERIES.

Etiology.—Though thrombosis of the femoral and axillary arteries belongs to the realm of surgery, as its chief symptom is lameness, still as its causes are entirely internal, and it is only a question of larger vessels, it may also be regarded as an internal ailment. Its origin lies, on the one hand, in an affection of the inner membrane of the artery (endarteritis), with deposition of fibrin-coagulæ upon the roughened surface and stenosis of the lumen; on the other hand, in embolic stoppage of this branch of the aorta by thrombi which have been floated thither, and have their source in the heart or in an aneurysm. The cause is occasionally to be sought in marantic thrombosis, which may occur in severe general debility (acute course). The condition is most frequently observed in horses, rarely in cattle.

Anatomy.—The aorta is most frequently found blocked with thrombi at its division into the two iliac or femoral arteries, and in the two pelvic arteries, less frequently in the axillary or brachial artery, and only in very rare cases in the lumbar artery. The end of the aorta and the root of the bifurcating arterial branch are mostly widened, the walls thickened, and atheromatously degenerated, the intima dull, fattily-degenerated, calcareous and covered with ulcers. Within is fixed a thrombus, which is usually stratified, organised and hard, of pale colour, and which more or less narrows the lumen, often leaving only a very small passage. The thrombus often grows forward into the aorta on one side, and into the arterial branch on the other, or adheres to the place of bifurcation. Sometimes the iliac or pelvic artery of one side is entirely filled by the thrombus. With this thrombosis a compensatory hypertrophy of the heart is not unfrequently developed as a secondary symptom. Also upon softening of the thrombus, emboli occur in the periphery of arteries in the extremities.

Symptoms.—The thrombi just described usually cause no signs of illness during rest, only after prolonged work in harness or saddle does the peculiar and characteristic aspect of the disease appear.

1. *In Thrombosis of the Femoral Artery*, which mostly affects horses, cattle more rarely, we notice first of all a gradually

increasing weakness of one or both hind-legs. The animal shows a characteristic intermittent lameness, generally on one side, strikes the affected hind-foot against the other, puts the toe only on the ground, or drags the foot behind, trembles, breaks down, and then lies for some minutes on the ground, struggling until quite exhausted. The breathing is extraordinarily accelerated, the heart-beat becomes rapid and jerky, the visible mucous membranes much injected, and the whole body covered with perspiration. The temperature of the affected limb is usually very low, and pulsation is often, if not always, absent from the tibial and metatarsal arteries. After a few minutes the horse rises, and its lameness by degrees passes away. Of importance for diagnosis is the fact that an attack can be experimentally produced by forced movement. On examining the location of the thrombus through the rectum, we find the aorta and its branching artery widened, thickened, unyielding, filled with a hard, elongated body, and generally without pulsation on the affected side. In rare cases the thrombosis is complicated with peripheral, venous embolism with gangrene of the limbs. The clinical aspect of an intermittent paralysis may be produced by pressure due to the presence of a sarcoma at the point where the aorta leaves the heart (Fröhner).

2. The less common *Thrombosis of the Axillary Artery* reveals itself in simple lameness upon the side affected. During movement the animal begins to stumble, straddles, continually knocks its toe, cannot lift its feet, but quite drags them along, trembles upon the affected side and finally rolls to the ground. We have, however, not noticed any disturbance of breathing, excitement of heart, nor signs of congestion. The animal soon recovers its powers for the time being.

Therapeutics.—Treatment of the thromboses as above described generally has but little effect, certainly internal treatment (iodide of potassium, alkalies) is almost useless. More effectual appears to be the massage of the thrombus by way of the rectum recommended by Colin and Bayer, viz., careful stroking and kneading of the plug, thus softening it and aiding its resorption; but there is a danger here, that some parts of the thrombus may be embolically carried away. Finally, by methodically exercising the animal, we may strengthen the cardiac muscles, increase the blood-pressure, and thus secure an improvement of its general condition. Absolute rest for several weeks has in many cases been known to

produce a very favourable result (resorption, or organization of the thrombus). On the other hand, in some cases we have seen the thrombus very much enlarged after such a lengthy rest, so that no opinion of real practical worth can be offered on this point.

Formation of Thrombi in the Heart.—This arises from disease of the endocardium, viz., in ulcerative valvular endocarditis, whereby weakness of the heart, with simultaneous stagnation of the blood-column, exercises a pre-disposing influence. The thrombi, which at first are small, only give occasion for general disturbance when they lead to the narrowing of an orifice. Symptoms of stenosis of the respective orifices then develop, with subsequent dilatation of the heart; emboli in the lungs and towards the periphery of the body also occur. A horse with thrombosis of the pulmonary artery suddenly showed such extreme dyspnœa that it had to be slaughtered (Käppel). These heart-thrombi, which usually adhere firmly to the endocardium, must not be confounded with blood-coagulæ in the heart.

WORMS IN THE BLOOD. HÆMATOZOA.

Occurrence.—Several varieties of worms are found in the blood of various animals, especially dogs, viz.: 1. Filaria immitis; 2. hæmatozoon subulatum, or strongylus subulatus, or strongylus vasorum; and 3. strongylus inflexus (found by Hering in seals and dolphins).

The most important of these is certainly *filaria immitis*, a round worm, said to have been discovered by Leydy, 1858, in Philadelphia, and identical with the filaria papillosa hæmatica of Dela'ond and Gruby. The fully developed filariæ attain a length of from 6 to 13½ inches and a thickness of 1 mm. They are to be found knotted together to the number of 50 to 75 in the ventricles, in the pulmonary artery, etc. Their embryos live in hundreds of thousands in the blood of dogs, and are on an average ¼ mm. long, by 5 micro-millimètres thick. Lewis found these filariæ embedded as knots in the walls of the aorta of an Indian dog. According to Janson, they are dangerous, owing to their wanderings throughout the body (thrombosis of the posterior and anterior vena cava, veins of the liver and kidneys, and of the lumbar, axillary and jugular veins); also owing to their closing the valves between the ventricle and auricle, *i.e.*, by twisting themselves around the chordæ tendineæ. In the case of a dog, Guittard found that the extremely thin wall of the right auricle had been pierced,

causing hæmorrhage into the pericardium. In the lungs, nodulous cysts containing the eggs of the strongy us vasorum were found. Von Rátz found the filaria immitis in the subcutaneous and inter-muscular connective tissue. Cadéac observed embolic pneumonic areas with the formation of cavities.

Symptoms.—However numerous may be the presence of filariæ in the blood, they usually produce no signs of disease for a long time, though in some cases they cause apoplectic death by the creation of thrombi in the heart, by stoppage of passages and even rupture of the heart. Emboli of their embryos in the capillaries of the brain appear also to produce apoplexy of that organ. Nephritis, cough, vomiting, pain, epileptiform convulsions, dyspnœa, emaciation, great feebleness, painful lameness of a hind-leg (symptoms resembling rheumatism), signs of lameness in the hind-quarters, hæmorrhage in the lungs and bowels (gastro-enteritis hæmorrhagica), and finally even appearances of rabies have been observed.

The course of filaria-sickness is chronic. We may differentiate between a *latent* stage, lasting for months or years without any visible signs of illness, and an *evident* stage (acute or peracute) with the symptoms described above.

Diagnosis.—The recognition of filaria-sickness during life is difficult, owing to the general character and multiplicity of the symptoms and their absence in the first stage. Diagnosis is nevertheless much assisted by the observance of two factors. First of all, the complaint, as a rule, occurs only in dogs which have lived formerly abroad (China, Japan). We find then, on careful examination of the blood, the characteristic embryos of filaria, $\frac{1}{4}$ mm. long by $\frac{1}{200}$ mm. thick. This latter examination ought therefore to be made with all imported dogs which show signs of emaciation, paralysis, etc. In one case which we observed, a dog brought up in Berlin was affected, but this was exceptional, and on dissection, no sexually ripe animals could be found, but only embryos.

Therapeutics.—Until the method of immigration of the filariæ is determined, no treatment is of use, and even then it can only be of prophylactic nature. The suggestion that they are transmitted by inheritance by breeding from affected dogs is very doubtful. Arsenic is said to have been given in Japan with good results to destroy the parasites in the blood.

History of Development.—According to Manson, the mosquito of the tropics in the New World, by absorbing human blood, becomes the intermediate host of the filaria sanguinis hominis. As soon as the filaria has penetrated from the mosquito's stomach through its body cavities to the thorax, its movements cease, and it begins to grow, passing through six stages of development. After the female mosquito has laid its eggs and perished in the water, the filariæ break through her skin, and play freely about in the water, so that men may be infected in two distinct ways, viz., by drinking such water, or by being bitten by mosquitos which have previously sucked blood containing filaria sanguinis hominis. In a similar manner we must imagine dogs to be infected. In Japan, where 50 per cent. of all dogs, especially those imported, yearly fall victims to the filaria immitis (Janson found it in 86 dissections out of 193), the opinion is general that the embryos discharged with the urine reach the rice-fields, where they collect in puddles, and are again absorbed by the dogs. According to Vryburg, filaria occurs in the aorta in 50 per cent. of all Indian buffaloes, but not so frequently in cattle. Hæmatozoa filaria attenuata are of very frequent occurrence in the blood of crows, and according to Leuckart are found in 80 per cent. of all crows. They have also been found in caterpillars (Mazzanti, Müller).

Eustrongylus Gigas was in one case found in the cardiac cavity of a pig (Fabretti).

Spiroptera sanguinolenta was found by Pease in the thoracic aorta of Indian dogs.

CHAPTER X.

SKIN DISEASES.

Introductory Remarks.—Skin diseases occur in many varied forms in all of our domestic animals, especially in dogs, horses and sheep, also in cattle, pigs, goats and cats, and are very common in poultry. The multiplicity of form in skin troubles has for ages led to the establishment of a great number of separate diseases (some of them falsely taken from human medicine), and to many different names, not all well chosen. Ring-worm, dry-rot, erysipelas, St. Anthony's fire, scabies, mange, greasy heels, scall, itch, scurf, were terms often applied indiscriminately to the same complaint, which have survived to the present time, and have caused more or less confusion. Although it would be better to discard these old names, yet in treating of skin diseases we cannot, for the sake of clearness, altogether dispense with them. It is, however, imperative to abandon some of them, and to try to group the great number of separate skin diseases under certain general heads. Thus we propose to treat several forms, which have hitherto been separated, from the stand-point of eczema, and follow the example set by Hebra in human medicine, who has very much widened the conception of eczema by including therein some apparently heterogeneous diseases. The expression "ring-worm," or "dry scab," we shall apply only to vegetable parasitic skin affections, as proposed by Gerlach, and the term "mange" solely to skin eruptions caused by mites or the acari of scabies.

A strictly scientific sub-division of skin diseases is much more difficult with domestic animals than with man, owing to their skins being covered with hair and pigment. For instance, diffuse or localised reddening of the skin can only be considered in animals whose skin, or portions of it, are free from

pigment. No system of diagnosis can be built up on the basis of pathological changes because the same lesions may occur in the most diverse skin diseases, and, vice versa, the same skin disease may in its course reveal the most varied pathological aspects. Thus, the term "scabby nodules" indicates no clinical unity, as nodules occur in a series of skin complaints. On the other hand, eczema exhibits several lesions (hyperæmia, nodules, blisters, vesicles, pustules, scabs and incrustation). It also does not seem opportune to divide skin diseases into acute and chronic, for many acute conditions of the skin, such as eczema, for instance, may often become chronic. A further division into idiopathic forms, or such as are caused by external injuries, and symptomatic, arising from internal morbid causes such as infectious disease, is also not unobjectionable, for some complaints, for instance urticaria (nettle-rash), a clinically sharply defined skin affection, may be produced externally (by insects) and internally (by strangles, swine erysipelas, nettle-fever, and pleuro-pneumonia contagiosa or murrain). Etiology is therefore by far the most important principle for subdivision in skin disease, but even this cannot always be adhered to, as in many skin affections their etiology is as yet unknown.

If we therefore adopt as most suited to our purposes the division into parasitic and non-parasitic skin diseases, we would first point out that the province of vegetable-parasitic skin eruptions is as yet very imperfectly fixed, and that the opinions of authors differ greatly as to the part which the various fungi play in the production of the several diseases, especially as vegetable micro-organisms may chance accidentally upon an already diseased skin.

After these preliminary remarks, we propose in the following chapter to adhere in the main to our subdivision into parasitic and non-parasitic skin diseases, the former of which we further divide into vegetable and animal parasites. The greater part of the symptomatic eruptions (variolæ, foot-and-mouth disease, etc.) arising from internal causes, as distinct from idiopathic affections, which we may also term "Exanthemata," and which "blossom-out," so to speak, from some internal disease, have been already treated among infective diseases in our second volume. Many skin affections such as erysipelas, phlegmony or cellulitis, panaritium, new growths upon the skin, etc., must be left to surgery.

General Symptomology of Skin Diseases.—In man the symptoms of diseases of the skin may be classed as either subjective or objective, but in the domestic animals it is only possible to describe the more important of the objective signs. The objective symptoms of skin affections are known as efflorescence or exanthema. They may be divided into primary and secondary, and, pursuing the classification customary in human pathology, the following elemental morphological forms are recognised :—

I. *Primary Exanthema.*—Of these, the most important are :—

(1) *Maculæ*, or marks, due either to anomalous conditions of the blood-vessels (red marks), or to anomalies of pigmentation (white, yellow, brown, or black marks). The discolourations caused by circumscribed or diffuse hyperæmia are known as *roseola* or *erythema*. Hæmorrhagic patches are described by the general term *purpura*, and, according to their form, are classified as *petechial* (spots), *vibices* (stripes), and *ecchymoses* (diffuse hæmorrhagic). Red marks in animals are less often caused by enlargement of the blood-vessels and angioma; such are *birth-marks*, *vascular nœvi*, and *telangiectasia*. Congenital deficiency in pigmentation is known as *albinism* or *leucopathia*; acquired deficiency, as *vitiligo* (white spots). The occurrence in the skin of pigment in abnormally large quantities, in the form of yellow, black, or brown patches is known as either *lentigo* (size of a linseed), *nœvus pigmentosus* (size of a shilling), or *chloasma* (diffuse), according to the size of the patches.

(2) *Nodules* or *papulæ* are elevations of the skin which occur in close proximity, and vary from the size of a millet-seed to that of a grain of linseed. They are due either to local inflammation and the formation of new tissue, or to the retention of secretion by the glands of the skin. The papula is to be distinguished from the vesicle by the fact that it contains no free fluid.

(3) *Vesicles.* A vesicle is an accumulation of fluid under the épithelium, varying in size from a pin's-head to a pea. It is caused by serous exudation from the capillary layer. Similar vesicles upon the mucous membranes, mostly the result of a coagulation necrosis of the epithelial cells, are known as *aphthæ*. Large vesicles are called *blisters*, *blebs* or *bu'læ*.

(4) The *Pustule* is a blister filled with pus. In other words, it is an elevation of the epithelium brought about by suppurative infection and change in the serous contents of the vesicle.

(5) *Blotch*, *nettle-rash*, or *urticaria* is a flat, mound-like cir-

cumscribed efflorescence, resulting from local serous exudation in the superficial layers of the skin.

II. *Secondary Exanthema.*—These are either brought about by the mechanical irritation of the skin (galling), or they are the further developments and ultimate stage of primary exanthema. The most important are: *Excoriation* (desquamation of the epithelium); *squamæ* (epithelial scales, sloughing of the epidermatoid cells in powder, bran, or flake form); *crustæ* (scabs and crusts of dried blood, serum or pus); *rhagades* (chaps, especially on the flexor surfaces in the form of cracks and fissures); *ulcers* (decomposition of tissue); and *scars* (new formation of connective tissue, covered with epidermis).

Anatomical Classification of Skin Diseases.—(Jessner and Schindelka).—I. *Functional abnormalities of the skin:* Pruritus cutaneus, derangements of the sweat and sebaceous glands.

II. *Abnormal distribution of blood:* Hyperæmia, anæmia, œdema (urticaria, prurigo), hæmorrhages.

III. *Inflammatory conditions:* (1) Inflammation of the cutis and sub-cutis; eczema, herpetic diseases, itch, animal parasites. (2) Inflammatory conditions without apparent exudation; herpetic diseases, pemphigus, foot-and-mouth disease, dermatitis, erysipelas. (3) Inflammatory conditions with considerable tissue disturbance: phlegmon, furunculosis, carbuncle, glanders, pseudo-glanders and ulcers. (4) Inflammatory conditions of the glands and follicles: acne. (5) Eruptions due to the action of drugs and fodder.

IV. *Granuloma:* Tuberculosis, actinomycosis, botryomycosis.

V. *Hypertrophy:* Ichthyosis, callosities, horny growths, warts, pachydermia.

VI. *Ulcers:* Of the connective tissue, of the epithelium, cysts.

VII. *Retrogressive disturbances of metabolism:* Atrophy, gangrene.

VIII. *Atrophy of the hair:* Alopecia, trichorexis.

IX. *Abnormalities of pigmentation.*

X. *Foreign bodies in the cutis and connective tissue of the sub-cutis.*

I.—NON-PARASITIC SKIN DISEASES.

Erythema of the Skin. Dermatitis Erythematosa.

Definition.—The simplest form of skin affection consists in hyperæmia of the papillary bodies and superficial papillæ of the skin, which is known as erythema of the skin and occurs sometimes diffusely, and at others in spots and locally (roseola). Such a condition is, however, only visible in domestic animals upon the unpigmented portions of their skin. We find it therefore most frequently in sheep, pigs, dogs, white-skinned horses, piebalds, as well as on the white parts of horses and cattle, about the head and extremities. The only perceptible symptom of erythema is a more or less localised or diffuse redness of the skin which, in contrast to hæmorrhagia (petechia, ecchymosis, purpura) can be made to disappear for a time by pressure with the finger. This redness is sometimes accompanied by itching (pruritis). If long continued, the erythema produces a more or less considerable desquamation of epidermis. In contrast to eczema there is no weeping.

Etiology.—Erythema of the skin occurs either independently or forms the introductory stage of other skin diseases. In the former case it generally soon disappears, leaving no pathological change behind in the skin. Its causes are :—

1. *Mechanical* influences upon the skin, such as pressure, friction, shearing in sheep, horses and dogs, whipping swine in transport, etc., so-called *erythema traumaticum.*

2. *Chemical irritants*, slight erosions, inunction of carbolic, tar, cantharides or tartar emetic ointments, or of black soap, sinapisms, etc. ; decomposition of urine on the skin near the place of discharge during paralysis of the hind legs ; insects' stings ; the effect of fungi upon the skin. This is known as *erythema toxicum.*

3. *Thermal influences*, excessively high or low temperature (first stage in scalding, burning or freezing) ; the effect of direct sunshine upon the non-pigmented skin (so-called sun-scald of horses). Known as *erythema caloricum* and *solare.* Schindelka observed *erythema neonatorum* in puppies and kittens.

Therapeutics.—Treatment is seldom necessary. Beyond removing the causes, we may apply as a relief for intense itching, fomentations of lead-water, or sprinkle a powder of

starch and oxide of lead, or a lead or zinc ointment; or, if the pruritis be very intense, apply a 6 per cent. solution of nitrate of silver.

Eruption produced by Buck-wheat (Fagopyrismus).—This is a peculiar form of erythema of the skin. although lying somewhat outside its simpler forms. It may run its course as true erythema, but as frequently represents a complication of erythema with erythematous inflammation of the skin (*i.e.*, inflammatory infiltration of the cutis or inflammatory œdema of the skin), or with vesicular, bullous, phlegmonous, erysipelatous or even gangrenous dermatitis. In general no strict line can be drawn between erythema of the skin and an erythematous inflammation. The original cause is to be sought in the eating of buck-wheat (polygonum fagopyrum) and of other polygonum species (polygonum persicaria), not only of the green buck-wheat, and especially at the time of flowering, but also of the grains, straw, stubble and chaff. Sunshine seems also to be necessary as a second factor. We can therefore best imagine the disease originating as follows: the parasitic fungi living on the buck-wheat, or their irritant products of metabolism, come in contact with the animal's skin, but can only affect it upon the non-pigmented portions, and then only when they are first made sensitive by the sun's action in producing erythema solare. Whether insects can produce this buck-wheat eruption in some instances (such fields are much favoured by bees), or whether some animals are peculiarly sensitive to the influence of the buck-wheat is uncertain. The sickness is commonest in white and white-spotted sheep, especially lambs and pigs; is rarer in goats and cattle, and very rare in horses which have unpigmented portions of skin. The more buck-wheat an animal has eaten, and the more it has been exposed to the sun's heat, the more violent is the outbreak. Black animals or such as are blackened over, do not contract it. The same, too with those kept in the stable, or pastured only in cool weather or fed in the shade. On the other hand, the eruption appears even if they have discontinued eating buck-wheat for eight to ten days, or even three to four weeks, and are then taken out into the blazing sunshine. On returning to the stable, the ailment soon vanishes, but returns on coming back to sunlight. In winter the eruption seems to be restricted to mere itching and burning.

The phenomena consist in intense redness (erythema) and inflammatory swelling (dermatitis erythematosa) of the head, especially the ears, face and eyelids, which may reach the inter-maxillary space and throat, accompanied by intolerable itching. The animals naturally grow irritated, shake their heads, rub themselves, run about restlessly, and even manifest extreme signs of inflammation of the brain, as in mania. Sometimes little blisters arise on the red and swollen skin the size of a lentil, or from that to a pea, which on bursting discharge a yellow fluid, and then heal, leaving a scab. This is the so-called head or pock erysipelas of sheep. We may observe in graver cases some disturbance of breathing (erysipelatous inflammation of the mucous membrane of the air passages), general feverishness, digestive trouble, as well as symptoms of brain affection: stupor, heaviness, rotary movements, spasms, great excite-

ment, etc., probably during the spread of the erysipelatous processes from the skin of the head to the meninges ; in such cases the ailment may even result in death. The epileptiform fits observed by Rabe and others in horses and pigs, without any accompanying skin affection, complicated with signs of vertigo, seem to indicate the presence in buck-wheat of some narcotic poison.

Treatment consists, as regards prophylaxis, in taking white sheep to graze on buck-wheat pastures on cloudy days or towards evening, or else serving the buck-wheat exclusively when under cover. When once the disease has shown itself, the animals must be driven without delay to a shady place or, better still, back to their fold. Local treatment is only necessary in severer complications, especially in erysipelatous inflammation of the skin of the head with cerebral and general symptoms. We should apply cooling bandages, wash the head with creolin water, olive-oil in combination with lime-water as well as anti-phlogistic remedies, such as tartar emetic, neutral salts, etc.

ECZEMA.

Definition.—Eczema may be best defined as a dermatitis, manifesting itself in various stages of development and intensity. In its nature and course, therefore, eczema corresponds entirely with inflammation of other organs, such as the mucous membranes, whence it was formerly regarded as "catarrh" of the skin. As there are various forms of one and the same catarrh of the mucous membranes, corresponding with the degree of inflammation, viz., serous, mucous and purulent, so, too, the clinical aspect of eczema is varied according to the acuteness of the inflammation and the nature of the affected portions of skin. These multiform outward appearances of eczema led formerly to the classification of a great number of apparently different and independent skin diseases under this name. This can be the more easily explained, seeing that the selfsame skin affection presents seemingly great differences according to the kind of animal or the locality, and that the simplest of such affections might be vastly changed in appearance by the animal rubbing, scratching or gnawing. Also on the further ground that identical causes might produce quite different aspects of disease according as the individuals varied in sensitiveness, or according to place affected, or the constitution, age, etc., of the animal. The classifying of so many separate forms of skin disease has been productive of such confusion and obscurity, that a reduction in their number is urgently needed, both for purposes of instruction and for consideration of the diseases from a common stand-point.

through all these stages. An early stage may pass directly into the last, or the eczema may long remain persistently in the same stage. We must not omit to mention that, owing to the pigmented or hairy skins of the animals, it is easy to overlook some of the rapid stages, such as the papular and vesicular.

General Treatment.—Remove the originating cause by cleansing or disinfection, as where this is not done other methods are frequently unsuccessful. The most important part of the treatment is local in character, directed immediately to the actual morbid condition of the skin, and with this internal remedies may be combined as purgatives, arsenic, etc. The local treatment should be regulated by the nature, extent and duration of the disorder; by the sensitiveness of the skin; by certain individual peculiarities; and by the species and breed of the animal to which it is applied. It is important to note that different treatment should be employed in acute and in chronic eczema. In acute eczema, palliatives and unguents should be used, such as neutral dusting powders, oils and salves, and all irritants, such as tar or soap, must be avoided. In chronic eczema, on the contrary, irritant and even corrosive remedies are indicated (tar, soap, nitrate of silver, sulphuret of potassium). Before the application of any remedy, accumulations of scabs or scales should be removed or softened by glycerine or wet compresses. The successful treatment of eczema depends to a large extent upon care, persistence and individual attention.

Classification of Eczema.—Varieties of eczema may further be classified according to the species of animal affected (horse, dog, cattle, sheep, swine). Schindelka distinguishes between an acute and a chronic form. The acute condition occurs in all the domestic animals and commences as erythematous or papular eczema. The form most frequently met with is moist eczema (e. madidans or rubrum), which is, as a matter of fact, the eczematous form at its worst, e. impetiginosum, crustosum and squamosum being degenerative forms. Acute eczema runs a course of 2 to 6 weeks; the periodic occurrence is very striking. The local changes are frequently accompanied by symptoms of general disturbance, such as fever, gastric derangement, irritability, swelling of the lymph-glands in the parts affected. The recurrence of acute eczema leads to a chronic condition. The symptoms of chronic eczema are similar to those of the acute form, but are more varied. Chronic eczema may appear as either a dry, scurfy, or as a moist, eruption. The dry form is most frequently met with among the domestic animals. It shows itself as a blue-red or brown-red discoloration of the skin, with a covering of

scurfy scales. The moist form is more rare (formation of scabs, with thickening of the skin). Chronic eczema does not affect the general health. It occurs principally on the parts covered by long hair (e. rubrum), or by short hair (e. squamosum). It makes its appearance in the case of horses at the joints and at the corners of the mouth, and is accompanied by thickening of the skin (e. tyloticum), and cracks (e. rhagadiform). It is more particularly met with upon the flexor surfaces of the fetlock, the carpal and tarsal joints, and the upper lip (due to nasal discharge) of horses; in the auricle (e. squamosum in horses, cattle, swine and sheep; e. madidans in the dog); upon the nipples of animals in milk (e. madidans and crustosum); *the scrotum of the dog (moist eczema)*; *the ischio-rectal* region (highly irritating e. squamosum in dogs, horses and cattle); the points of junction of the skin and mucous membranes (eyelids, nose, vagina). Schindelka and others describe certain peculiar forms of eczema as seborrhagic eczema in horses and dogs (round scales or scabs, covering reddened or moist skin; " mane-scab " " tail-scab "); and sycosiform eczema in horses (pustulous inflammation of the hair follicles; folliculitis in the form of knots, in the eczema which occurs on parts covered by long hair, such as the fetlocks).

A.—THE VARIOUS KINDS OF ECZEMA IN DOGS.

Occurrence.—Eczema is the commonest of all skin affections, and one of the most frequent ailments of the dog. According to our experience at the Berlin dog-hospital, one-twelfth of all dogs suffer from it (5,462 cases of eczema among 70,000 sick dogs, in the years 1886–1894). It is mostly restricted to certain parts of the body and usually occurs along the back, beginning at the root of the tail or on the neck, and spreading thence both lengthwise and sideways. Other favourite spots are the outer surfaces of the extremities and the rump. It appears more rarely on the lower breast and belly, on the knee-folds or elbow-joints or upon the protuberances of the astragalus, seldom upon the head. Old dogs which live much in the house are most prone to it, as well as young, tender-skinned, pampered and therefore fat ones. As regards race, pugs, fox-terriers, Danish dogs, mastiffs and the Leonberg breed seem to be most susceptible.

Causes.—Eczema is usually set up as the direct result of some external irritant acting upon the skin, such as dust, mud, fleas, lice and insects, or of repeated friction and long-continued pressure on certain portions of skin, etc. The favourite place for eczema, the back, is specially exposed to dirt, dust and fleas. Washing or rubbing with soft soap (green or black) often causes eczema in dogs. The fact that an external

cause can in many cases not be assigned has led to the presumption of an internal origin. In this way injurious food, digestive disorders, polyæmia and general bodily debility have all been cited. These factors can be regarded only as pre-disposing causes, and a direct local irritation must always be presumed.

Symptoms.—The symptoms of acute eczema in dogs are so varied that a description suited to all cases is impossible. The appearances differ according to the stage of the disease.

1. In the initial stage, which usually passes unobserved, we see bright red spots, the size of a pin's head, upon the non-pigmented parts of the skin, which quickly grow darker, forming a nodule or pimple in the centre of each, while the hyperæmic back-ground increases, the spots gradually run together, so that the affected sk n becomes spongy, turgescent, and of a higher temperature. The hair round these pimples stands on end in tufts; a slight touch of the skin sets up itching, and a harder touch causes pain. As the pimples soon disappear we often perceive only the aspect of erythema or of erythematous inflammation. In this stage the eczema may heal, or may remain some time stationary, though this is rarer. Its condition is often much chang d by rubbing and gnawing on the part of the dog, so that the pustular and scaly may follow immediately on the papular stage.

2. If the eczema runs its regular course, small blisters which are sometimes isolated, at others grouped, and are at first full of clear fluid, develop from the pimples. As they are seldom larger than a hemp-seed and very readily burst on being scratched, they are difficult to find on a dog's hairy skin and are often unobserved. They partly dry up and are then covered by a slight scab, some burst or are scratched open and then form minute foci of inflammation, above which the hair becomes loosened and falls off. If the eczema heals when in this stage, the fluid in the small vesicles dries up into small crusts, beneath which the inflammation recedes and the skin is restored. Upon the darke: parts of the skin he position of these blisters is shown for some time by the patch being poorer in pigment and devoid of hair.

3. The eczema is more frequently inclined to spread superficially, and by confluence of many blisters we obtain spaces without both outer skin and hair, and which are very much

reddened and wet (eczema rubrum), covered with serous, sero-fibrinous and even purulent exudation, exceedingly sensitive to touch and readily attack the adjoining sound parts (corrosive rash, eczema exedens). Or, in consequence of the animal's rubbing, the eczema progresses to an intense purulent and hæmorrhagic dermatitis. The cure of eczema rubrum occurs by drying-up of the exudation (scaly stage) and regeneration of the subjacent epidermis from its intact peripheral edges.

4. Not unfrequently the vesicles change into pustules, and we then have a pustular eczema. The blisters enlarge and their contents grow dull and become purulent. Like the blisters, the pustules also stand isolated or are spread in groups, and are also not easy to find on the hairy skin. They often run together, producing purulent patches of various size, when the hairs bristle, stick to each other and are glued with matter into stiff tufts, become felted, are easily pulled out or fall out of themselves. The skin thus laid bare is covered with a yellowish to greenish-yellow, sticky, creamy pus, is very painful and readily bleeds when touched, the skin itself becoming thicker (eczema impetiginosum). Healing follows by drying-up of the purulent exudation into scabs and crusts, beneath which suppuration may continue for some time. In rarer cases the process goes deeper and develops to a phlegmonous inflammation, as well as to ulcerous destruction of the cutis, the latter especially when the parts have previously been much rubbed or gnawed. Hairless places then remain behind, the skin being indurated and thickened, and covered with abundant scales. Similar localised purulent inflammations of the skin occur, without any previous eczematous blisters or pustules, as a direct result of strong mechanical, chemical or thermal irritation.

5. Eczema may in all stages become chronic. The most important factor here is to prevent the animals from rubbing the places affected. Generally the changes left behind after severe eczema cause a predisposition to subsequent acute outbreaks, which are commonest in the summer months, because the afflux of blood to the skin is then greatest. The changes produced in the skin by chronic eczema consist in persistent hyperæmia, higher temperature and thickening. Thus the skin of the back on affected portions often feels three or four times its usual thickness. The surface, too, of the thickened parts seems smoother and more shiny, and often a permanent and considerable proliferation of epidermal cells takes place, which then fall off as white to lead-grey scales (eczema squa-

mosum). Gradually the skin becomes harder and dryer, rigid; if laid by hand in folds, it remains so for a long time; the hairs bristle in all directions, are thin and look dry and pinched, or are even lacking entirely or curled like wool (compression and atrophy of the hair-papillæ and their vessels by new growth and retraction of the connective tissue). Such patches of skin often wear a granulated aspect, whereby the hypertrophic papillæ of the skin protrude in the form of thick papillary proliferations above the outer circle.

The general condition of animals during eczema is as a rule unaltered beyond continuous excitement and unrest and a frequently manifested and extreme thirst. In cases of long duration, however, or with repeated relapse, gradual emaciation results, caused by the long-continued irritation, the increased waste of fluid and bodily heat, as well as the persistent reflex excitement transmitted from the skin to the entire nervous system, which in feeble or very young animals may develop to cachexia and death. Acute eczema may last from one to three weeks; the chronic form for months and indeed, with shorter or longer remissions, even for years, and is frequently incurable.

Differential Diagnosis.—Eczema may be mistaken first for a purulent inflammation of the skin caused by a wound, excoriation, erosion, scald or frost-bite; also for true herpes, especially herpes tonsurans, and finally for an acarus eruption or mange produced by mites.

1. *Traumatic dermatitis* exhibiting localised, reactive processes which are accompanied by suppuration or detachment of ichorous scurf or scabs of dry gangrene, etc., beginning with gangrenous destruction of a portion of skin, and secondary to which a suppurative, granulating dermatitis occurs. The papules, vesicles and pustules characteristic of eczema are entirely absent. Since these latter are occasionally lacking also in eczema, a differentiation may at times be difficult. Eczema frequently changes into traumatic, suppurative dermatitis as a result of persistent rubbing by the animal.

2. *True ringworm* (*tinea tonsurans*) may generally be distinguished by the rounded shape of the eruption, by frequent absence of all papules, vesicles and pustules, by itching, by its quality of being transmitted and finally by microscopical examination.

3. *Follicular mange* occurs mostly on the head and legs,

is infective, has its own typical morbid aspect, and is much more difficult to heal than eczema, if not incurable.

4. *Sarcoptic mange* occurs chiefly on the soft-skinned and less hairy parts of the hypogastrium, on the lower chest, the inner surface of the thighs, about the elbows and at the base of the ears ; it is contagious and usually accompanied by very intense itching. The clinical aspect does not always suffice for diagnosis, for both follicular and sarcoptic mange may much resemble a non-parasitic eczema, when only the microscope will enable a positive diagnosis to be made.

Therapeutics.—The treatment of eczema varies both according to stage and individuality. We must first seek to remove the causes. In most cases it would heal spontaneously if the abnormal irritation caused by the itching could be corrected. With most animals this is the chief difficulty. In many dogs the pruritis is so intolerable, and the attempts of the animals to rub themselves are so ingenious, that it is found impossible to secure this first condition of speedy recovery, in spite of muzzles, bandages, collars, boots, etc.

1. In the *lighter cases and earlier stages* of eczema (erythematous, papular and vesicular) antiphlogistic, emollient, anodyne, and protective dressings should be applied. We use either a simple zinc-salve or a freshly made lead ointment, especially Hebra's salve in its newer form: equal parts of litharge and paraffin ointment. As a protective powder for dusting on which will remain a long time lying between the hairs, especially on the ridge of the back, we recommend a mixture of zinc-oxide and starch (1 to 4). The otherwise excellent white precipitate ointment (unguent. hydrargyr. album) must be very cautiously used on dogs, owing to the risk of their licking it off.

2. In *excessively weeping or impetiginous* eczema we have long used with great success a 6 per cent. watery or spirituous solution of nitrate of silver, which, after first washing the weeping or suppurating place, we lay on with a brush, when a slight dry eschar or slough is formed, beneath which the healing process rapidly occurs. Among ointments we use most frequently a nitrate of silver salve (1 : 10–20 paraffin ointment); tannin ointment (1 to 9) and creolin-zinc salve (1 to 9). Besides these, dusting with absorbent desiccant powders acts excellently in persistent weeping. We prescribe, for instance, a mixture

of dermatol and starch in the proportion of one to ten; or of creolin with boric acid (2–4 : 100.)

3. For *chronic eczema* our chief remedies are tar and creolin. The former should be used either in solid form or as tar-spirit or in the form of a tar liniment in combination with soap potash, which latter is especially efficacious in chronic impetiginous eczema of the back. It is wise to clip off the hair before application and to protect the places afterwards by a covering of leather or some effective bandage to prevent scratching and licking (carbolic poisoning). Leave the tar on for four to eight days, and then remove the dry scab as tenderly as possible; if needed, repeat the inunction. Tar liniment has the advantage that it can be somewhat easily removed by bathing. Instead of tar we have for many years used creolin, which possesses the healing qualities of tar, with but slight risk of poisoning; viz., a creolin liniment (creolin, potash soap of each 3 oz., spirit 1½ oz.), creolin (1 : 10–20) and creolin ointment (1 : 10–20 paraffin ointment). For eczema squamosum we also use either the tar or the creolin liniment as above, and the tar or creolin spirits (1 : 10–20). Among other remedies we have also used with advantage chrysarobin (chrysarobini 1 to 4 scr.; unguent. paraffin. 5 drams), oxynaphthalin (1 : 10) as well as ichthyol. As an excellent astringent Trasbôt recommends iodine-glycerine (tincture of iodine and glycerine (1 : 4). To relieve irritation and promote scaling, Schindelka advises sapolan in the form of a paste (sapolan., vasel. lanolinat., zinc. oxyd., amyl. āā m.f. Pasta); and in chronic seborrhoic eczema in dogs he recommends sulphate of potassium baths.

These by no means exhaust the list of remedies used and recommended for eczema in dogs. We might mention also naphthol, anthrarobin, resorcin, naphthaline, pyoctanin, dilute nitric acid (1 : 5–10) the oxy-naphthalic acids, etc., but to enumerate them all would lead us too far. Among internal remedies for obstinate chronic eczema we would mention arsenic, which we have often used with good effect. It is usually given in the form of Fowler's solution, 5 to 10 drops at once, for weeks together. At the beginning of recovery, good may be done during certain stages of the malady by the use of laxatives, as then the drying-up process proceeds more quickly, especially in very weeping eczema. We have never noticed any good result from special dietetic treatment, such as withholding flesh-meat. Bär (in 1902) found micro-cocci in specimens of the skin of a

dog affected with eczema rubrum, and believes these to be the specific cause of eczema and is said to have propagated them successfully. For this reason, he treats eczema with disinfectant applications after previously cutting away the hair. He has found a 2 per cent. solution of creolin employed once or twice daily, very efficacious, but in obstinate cases he used ointments (zinc, lead, tannin). To conclude the treatment, he recommends an immersion bath containing 5 per cent. of sulphuret of potassium.

B.—ECZEMATOUS SKIN DISEASES IN HORSES.

General Remarks.—The non-parasitic skin diseases of horses are for the greater part purely eczematous in their nature. If we bear this fact in mind, an understanding of the many forms of dermatitis described as special eruptions in horses will be greatly facilitated. It will render much of the old nomenclature superfluous, as an aid to comprehensive study of these skin diseases. We include among eczemata the following skin affections, which have hitherto been regarded more or less as independent forms of eruption :

1. As *papulo-vesicular eczema* are to be regarded : such periodic eruptions as herpes or tetter, and also those skin diseases described as summer-mange, saddle-mange, heat-eruption.

2. As *squamous, chronic eczema* we must consider in the horse, as well as in the dog, pityriasis and psoriasis.

3. The so-called mane and tail " mange," as well as the disease of the hair known as plica Polonica, are a *chronic and frequently pustular, scaly eczema.*

4. The malady known as greasy heels in the bend of the pastern, as well as that called mallenders and sallenders in the bends of the carpal and tarsal articulations, both represent a *localised eczema,* which *at first* is *weeping and later becomes scaly.*

I.—PAPULO-VESICULAR ECZEMA OF HORSES.

(*Herpes, Lichen, Heat-Pimples, Summer-rash, Summer-mange, Saddle-mange.*)

Etiology.—The papulo-vesicular form of eczema in horses is usually restricted to certain parts of the body. More rarely the eczema is universal and spread over the whole body,

as in the so-called heat-rash in summer (summer-mange). Those parts which sweat most freely are more easily attacked, because more subject to mechanical action, viz., the head (bridle), the sides of the neck up to the shoulder, the parts on which the collar and harness rest, the back and sides of the breast (saddle, belly-band and traces) and the croup (breeching, tail-strap). Besides these external factors, youthfulness, a fine and tender skin, as well as the season for change of coat, may all be predisposing influences. In isolated cases, eczema appears in the neighbourhood of wounds, after the continued application of irritant antiseptic washes (lysol, creolin). In rare instances, it has been observed at the pastern after neurectomy.

Symptoms.—The most characteristic feature in papulovesicular eczema is the presence of nodules which arise on the spots named in great numbers, irregularly grouped, and at first can be detected only by stroking the hand over the part. In size these nodules vary from a millet-seed to a pea. Their consistency is at first somewhat soft, but they become afterwards harder and firmer. The skin round about them is slightly higher in temperature, and may even appear swollen. If laid in a fold and pressed lightly, it is found to be more sensitive. The hair upon these pimples is bristly. During the stage of efflorescence and of healing a moderate degree of itching is present, but not at the height of the disease. In the later stages the tops of the nodules are covered with small scabs, owing to a serous exudation beneath the epidermis (*vesicular stage*), whereby the pimples themselves become smaller and harder. These little scabs glue the hair above them into small tufts, which afterwards fall off, or are rubbed off, with the scabs. This leaves small, hairless patches behind, which on the unpigmented skin are slightly reddened, and on the pigmented parts covered with light-grey scales of epidermis. This scaling and desquamation of superficial epidermis may, when exudation is abundant, be very extensive (so-called scaly rash). If the inflammatory process has gone very deep, lighter, thin-skinned bald places (so-called scaly nodules) remain upon the darkly pigmented skin.

Treatment.—The eczema just described is harmless and usually heals spontaneously after its various stages have been run; the hair also grows again by degrees upon the bald patches. Treatment is, therefore, not generally needed. If it be

required, we should endeavour to soften the scabs and scales with glycerine or pure fat, or to stimulate the growth of the hair by rubbing in spirituous remedies. Spirits of tar and of creolin are the best for this purpose (1 : 5–10), or dilute tincture of cantharides (1 : 10 spirit) may be employed. Ichthyol is also recommended. In rare cases the treatment is very tedious, or the eczema, particularly when universal, defies all remedies: The causes of this incurable eczema, enduring for months and years, is not known. It is very difficult in such cases to overcome the continued irritation, which manifests itself in constant rubbing and chafing, as well as by great restlessness, especially at night. Imminger recommends the following ointment: sulphur, 50; mercury, 5; cantharides, 20; vaseline, 400. Müller finds that irritation is relieved by daily bathing of the parts with a 1 per cent. solution of permanganate of potash.

Sweat-eczema on the Back in Army Horses.—Steffens, Bartke, Grannulich, Hennigs, Kalkoff and others are of opinion that, since the introduction of the new military saddle, sweat-eczema, which was rarely observed with the old pattern, has become comparatively common. It appears almost exclusively after violent exercise upon hot summer days. It does not affect the part covered by the saddle itself, but occurs in the lumbar region, at the cantle of the saddle, along the edge of the saddle cloth. It is partly the result of irritation by sweat mixed with dust and grit, and by friction of the saddle-cloth.* The principal manifestation of this form of eczema is the occurrence of nodules ("heat-knots," "heat-pox"), accompanied by œdema and tenderness of the skin. *Treatment* consists in washing with creolin and water, Burow's solution, or alcohol, or with warm solutions of permanganate of potash, corrosive sublimate, nitrate of silver, or pyoctania. Priessnitz compresses are recommended. As a preventative measure, the saddle-cloth should be folded shorter, and the baggage carried behind should be lightened.

Winter Mange.—Under this name, Warncke describes a papulo-vesicular eczema in horses, which appears in the winter upon the flexor surfaces of the hocks, the inner surfaces of the thighs, at the root of the tail and under the mane. There is no irritation, but the disorder is very obstinate; it is related to summer mange, but Warncke does not consider it identical with any known form of eczema.

Prurigo.—Under this heading we indicate a papular, chronic and violently itching skin eruption in man, which attacks especially the inner sides of the legs, beginning almost exclusively in early childhood, and being obviously inherited. After lasting for years, it leads finally to

* A dirty saddle-cloth has been known to cause this and also ringworm in horses, and by its use on other horses the infection has been spread.

characteristic permanent changes of skin (thickening and pigmentation). Prurigo can seldom be cured, and many succumb to the prolonged intense itching, constant excitement and gradual exhaustion. This disease, which can be defined with the utmost precision in man, is said by most authorities to occur also in our domestic animals, when it is designated "itching eruption." But in our opinion there are no true grounds for assuming the existence among domestic animals of any complaint corresponding to the prurigo of man. The aspect ostensibly observed in various animals (horse, dog, cattle, sheep) does not agree with prurigo of man either in its causes or its phenomena and course. Indeed, we can find nothing that is characteristic in the descriptions of itch-eruption in animals. We have never seen a case of true prurigo in any animal, and are of opinion that a small part of those described as prurigo are nothing else but papular eczema, the descriptions exactly agreeing therewith; and that the major part, especially the chronic and obstinate cases, should be classed as mange. We have, therefore, not included prurigo among the skin diseases (dermatosis) in the interest of greater uniformity of treatment.

Pruritus, or pruritus cutaneus (itching of the skin), is the name given to a functional derangement of the nerves of the skin, characterised by violent itching, and which is not attributable to any local anatomical condition, primary eczema being invariably absent. In human pathology it is customary to distinguish between general and local pruritus. The condition in man results either from nervous derangement or from general conditions of ill-health, such as diabetes, nephritis, jaundice, senile decay, digestive disturbance, etc. According to Schindelka, pruritus is universally prevalent among the domestic animals. Its actual cause is at present unknown, but it is frequently possible to prove its connection with severe general or local disease (*mal de coït*, and icterus in horses; chronic digestive disturbances in horses and cattle; chronic nephritis, constipation, senile decay in dogs; foot-rot in sheep; trichinosis in swine). Schindelka states that local pruritus frequently occurs, without any apparent cause, at the tip of the tail in dogs. It appears in rabies, and also, in isolated cases, after mange. Schindelka saw a dog, attacked with severe local pruritus after mange, tear so violently at its fore legs with its teeth, whimpering all the time, that pieces of the flesh gangrened and afterwards dropped off. In the same category with pruritus, Schindelka classes the irritation of helminthiasis in dogs and the itching of the nose which occurs in the diseases caused by œstrus larvæ in sheep. He is unable to report any appreciable results from local treatment, though he has been able to obtain temporary relief by rubbing in salicylic acid, carbolic acid, or tar. In the only case which has come under our (Fröhner) notice, that of a horse, the irritation was in the left lower jaw. It had continued for 2–3 months, and was so violent that any attempt to prevent the animal from rubbing the place, by tying him up, etc., gave rise to a state of excitement verging on madness. The cutis and subcutis at the affected part were inflamed and swollen; there was necrosis and an ulcerous surface, penetrating into the sub-cutis, as large as a man's hand. Examination showed no signs of general or local disease, neither were mange-mites present, and the origin of the pruritus remained unexplained. Cocaine and caustic remedies (mercuric chloride in spirituous solution,

nitrate of silver) were both employed without result; antiseptics (creolin, tar) only served to increase the irritation. Finally, boracic acid and acetate of alumina gave some relief, but did not effect a cure.

2. CHRONIC SQUAMOUS ECZEMA OF HORSES:

(Bran and Scale Eruption, Hunger-mange.)

Etiology.—Squamous eczema is treated in some text-books as an independent skin disease under the names of "scale and bran scabies," and incorrectly identified with psoriasis and pityriasis of man. The most diverse forms of eczema may obviously become squamous during a prolonged course, thus giving the idea that they are peculiar forms of dermatitis. Their causes are, therefore, in general the same as those of eczema. Dirt and lack of attention to the skin, together with bad nourishment (so-called "hunger-mange"), seem especially to favour the disease. Of the operation of fungi nothing certain is known.

Symptoms.—The most important morbid phenomena of squamous eczema consist in an accumulation of small scales, like flour or bran, upon the skin, beneath which the latter suffers chronic change, viz., thickening, discolouration and induration; there is usually only slight itching. Such accumulations occur mostly about the head, especially over the eyebrows, upon the inner surface of the muscles of the ears, on the neck, base of the mane, shoulders, elbows, haunches, root of the tail and the flexor surface of the carpal and pastern joints. In these latter cases squamous eczema is designated as a form of mallenders and greasy heels. It is a harmless skin affection, with a chronic course continuing possibly for months and years: It occasionally heals spontaneously.

Therapeutics.—Treatment consists in careful attention to the skin, softening the scales with green soap, or application of tar or creolin preparations (salve, liniment, spirits). Tar and creolin seem to change the chronic inflammatory process into an acute one, which rapidly heals. Chrysarobin and ichthyol ointment has a precisely similar action (1 : 10).

Psoriasis and Pityriasis.—There is no ground for designating scale and bran scabies by these names with reference to the same diseases in man.

1. *Psoriasis in man* is an infiltration of the papillary body, with excessive development of horny substance on the superincumbent epidermis, whereby at first red nodules (*psoriasis punctata*) with horny, asbestos-like scales (*p. guttata*) appear, which attain later the size of a crown piece (*p. nummularis*), even coalesce, and may cover considerable areas: These patches often heal first from the middle, leaving ring-like marks, or, if many run together, resembling garlands (*p. annularis and gyrata*). Favourite spots are the inner sides of the elbow and knee-joints, the hairy scalp, the forehead and the ears. We have never seen any skin disease like this in our domestic animals.

Pityriasis in man is an extremely rare skin affection, which displays redness and scurfiness of the skin, sometimes over the whole body, and leads to atrophy and even gangrene of the skin, emaciation and death. Schindelka observed an eczema in pigs which he held to be identical with pityriasis rosacea.

3. CHRONIC IMPETIGINOUS ECZEMA ON THE LONG-HAIRED PARTS OF THE HORSE.

(*Mane and Tail-Scall or "Mange," Plica Polonica.*)

Occurrence.—We meet with eczema in its various stages, particularly the more advanced ones, such as the weeping and pustular, very often in horses upon those parts of their bodies which are covered with long hair, chiefly upon the edge of the nape of the neck (mane or neck-scall), on the tail (tail-scall), and more rarely on the head (head and lip-scall). The hair also participates secondarily in the consequences of this inflammation and disturbance of nutrition, and either sticks together and atrophies (elf-locks, plica Polonica), or else largely falls out (rat-tail).

Etiology.—The causes of impetiginous eczema upon the long-haired parts of a horse's body are purely local. First comes the uncleanliness and defective care of the skin, which permit the accumulation of dust upon these parts (that of hay, for instance) and of other irritating matter, and which afford especially good quarters for numerous parasites (lice, hair-mites). Eczema is consequently most common in bad and neglected establishments, and in those lands where the proper care of horses is purposely neglected, owing to certain mistaken or superstitious prejudices, as in Poland, Russia, Tartary ("Polish tuft"). On the other hand, excessive attention may lead to eczema, *e.g.*, too frequent washing of mane and tail with soap; when the soap is not always quite washed out

again, it may set up the inflammation by repeated and continued irritation. A prolonged state of wetness, after persistent rain or too frequent washing, may set up a maceration of the epidermis beneath the thick hair by the consequent warm moisture, as well as a decomposition of the normal skin-secretions (sebum, sweat), and may thus become a direct cause of eczema, like the rain-rot of sheep. To this the skin in such spots seems to be especially pre-disposed, being fine and tender, because well protected. Previous inflammatory processes in these parts often leave some after-effect, which predisposes to their recurrence (eczema squamosum). To what extent bacteria, and more particularly the pus bacilli, are to be regarded as the immediate cause of impetiginous eczema, or whether their influence is only secondary, is very difficult to determine. In one case, that of an epizoötic occurrence in a livery stable of purulent eczema of the tail, Caspar asserts that he was able to identify the streptococcus pyogenes as the primary cause of the condition.

Symptoms.—The incipient forms of eczema upon the long-haired parts naturally remain concealed, especially when little attention is paid to the mane and tail. Only when the products of inflammation have gathered and decomposed and the complaint is already far advanced, does the owner notice the altered look of the hair and the persistent and violent rubbing and gnawing of the horse. The changes are then mostly as follows :—The skin at the root of the hair shows over a larger or smaller area a coating which is sometimes serous, at others purulent, or bloody or finally scabby, and which has replaced the epidermis. Occasionally blisters and pustules may be observed. The hairs are stuck together by a sweaty or fatty matter, which is greasy, sticky, dirty and malodorous, often baked together, as it were, into balls and lumps as high as the hand, or felted into tufts and plaits (elf-locks, plica Polonica). In time the nature of the hair becomes changed by the spread of the inflammatory process to its papillæ. It grows thinner, curls more easily, and in this weakened state, felts and tangles more readily. In places which the animal can easily rub or gnaw, such as the tail, the hair becomes atrophied and falls out; and in course of time sclerosis of the skin sets in upon such spots, with subsequent depression of the hair-papillæ, ending in permanent loss of hair (rat-tail). The skin consequently grows thicker, more brittle and harder, and then passes into the chronic inflammatory stage of scaly eczema.

The duration of the process can in this manner become very tedious, and its cure difficult if not impossible.

Treatment.—In addition to thorough cleansing with soap, this consists in drying the weeping and impetiginous places. The better to do this, cut off the hair if possible close to its roots. Then apply to the now accessible skin a liniment either of creolin or tar, or else astringent or desiccant powders (iodoform with powdered oak-bark); or, finally, a six per cent. solution of nitrate of silver. The latter has a tendency to discolour light hair, to which fact attention should be called before using it. Salicylic acid (2 per cent. solution in alcohol) and pyrogallic acid (water solution) are recommended by others. Imminger employs an ointment composed of sulphur (50), mercury (5), cantharides (20), and vaseline (400). It is generally a good while before a complete cure can be secured.

Elf-locks.—In the foregoing we have regarded the so-called "Elf-locks," plica or trica Polonica, as a part-phenomenon of weeping and impetiginous chronic eczema, wherein a diseased and changed condition of the hair is produced by spread of the inflammation from the skin to the hair-papillæ. This is evidently only a casual result, so we wonder at the importance attached thereto in the older text-books. At first it was actually looked upon as an enzoötic malady, with which at the same time was combined the idea of a "critical" sign. Its appearance was said to bring relief to an internal complaint; its removal to act fatally! Spinola and others suppose "Elf-locks" to result from a constitutional disease. Haubner classes it among the effects of nutritional disturbance, while it has been ascribed by others to the agency of fungi or bacteria. Haselbach has recently described it as occurring in 10 per cent. of all horses in Poland, and considers it to be a disease of the hair in which a sticky fluid is excreted from the stems of the hair! The older theories were very crude, and, to make confusion worse confounded, they distinguished between a "true" and a "false" disease. By "false" Elf-locks is meant a simple matting together of the hair by dirt or other extraneous substance, such as bearded plant-seeds, thistles, burrs. The condition, however, whether "true" or "false" is one of slight importance. This has long been recognised in human pathology where the disease takes the form of a matting together of the hair combined with chronic moist eczema of the skin of the head.

4. ECZEMA OF THE FLEXOR-SURFACES OF THE LOWER JOINTS OF THE EXTREMITIES.

(*Eczematous Greasy Heels and Mallenders.*)

Occurrence.—Special names have always been given to localised eczemata on the flexor surfaces of the joints of the

extremities. The most important of these is the weeping eczema or eczematous greasy heels occurring on the pastern, which, in distinction to the pock-exanthema of horses, or so-called "dirt-mange," which also attacks the pastern, and the other surgical forms of greasy heels, is more exactly described as "gaping" or "dirt" greasy heels. Eczema on the flexor surfaces of the carpal and tarsal joints, or so-called mallenders, is much less common than greasy heels, because these joints are higher up, and therefore better protected against injurious material from the ground. Its course usually takes the form of chronic squamous eczema. For the other varieties of greasy heels—the erysipelatous, gangrenous and verrucous—consult the text-books on surgery.

Etiology.—The causes which produce greasy-heels operate both directly and indirectly. The flexor surfaces of these joints possess a peculiar pre-disposition to eczema, by reason of the constant displacement and formation of folds in the skin at every movement. Contributing causes are, grit and dust, from dirty roads or dirty stables; damp and wet, in rainy weather or during a thaw; and cold. Eczematous greasy heels are therefore observed more frequently in winter than in summer. Also the hind-feet suffer oftener than the fore-feet, being more exposed to the action of dung when in the stable. Prietsch noticed the occurrence of greasy heels after sprinkling salt on the tram-lines in winter; but Siedamgrotzky, Born and others, maintain that such sprinkling, according to their observations, did no harm in this way. Clipping the fetlock, which is particularly well-developed in low-bred animals, and which forms a natural protection from dirt to the fetlock joint, and cutting away the hair from the pastern, have been proved by experiment to contribute to the development of eczema by exposing the flexor surface to the action of dirt, damp and chill. The pus bacilli are, in many cases, the primary cause of eczema. But it is frequently difficult to determine whether micro-organisms are the originating cause of greasy heels or whether they are merely a contributing factor.*

Symptoms.—Mallenders and eczematous heels run an acute course, the symptoms developing in a very typical manner.

* Washing the legs and feet, especially in cold weather, and leaving the water to dry on is another predisposing cause.

First the skin is red, swollen, painful and warmer to the touch (erythematous stage), and small blisters soon make their appearance (vesicular stage), which burst and discharge a watery, yellowish fluid which is at first odourless (moist stage). Owing to constant movements, the swollen skin assumes thick folds, upon which fissures and cracks (so-called rhagades) readily form, become inflamed and quickly covered with dry scabs. The hairs are stuck together, or are bristly and readily fall out. The movement of the affected limb is stiff and awkward.

Although the lesions of mallenders and sallenders at this stage either heal or change to a squamous chronic form, with marked thickening of the skin and tendency to acute relapse, yet eczematous greasy heels, if long continued, may produce very serious changes in the fetlock. The secreted fluid decomposes upon the outer skin and macerates the epidermis. We then find a discoloured, greasy or pulpy secretion with a putrid smell, which, owing to its irritant properties, causes ulcerous loss of substance upon the skin, together with chronic thickening. Thus there arise upon the fetlock extensive sores covered with granulations which tend to become nodular or grapy (so-called "grapes"). The hair is at the same time erect and bristly. This thickening of the skin may, in the course of months and years, develop into true elephantiasis, in which case a chronic and permanent squamous eczema persists upon the outer skin (so-called squamous grease). Sometimes the leg is swollen as high as the elbow or thigh. "Thrush" is also a frequent accompaniment of chronic greasy heels. Finally, through the introduction of specific material, the eczematous greasy heels may develop into the erysipelatous and gangrenous forms.

Therapeutics.—The treatment of these conditions varies according to the stage of the disease. At first cleanliness and the application of a simple desiccant powder (such as oxide of zinc and starch) or powdered oak-bark are sufficient; some prefer zinc or lead ointment. In later stages stronger measures must be adopted. First, we must endeavour to restrain secretion and decomposition of the discharge by desiccant, disinfectant and astringent remedies. For this purpose creolin and tar liniments are valuable, and desiccant powders, such as tannin, tannoform, amyloform, glutol, xeroform; turpentine dressings; alum baths; sublimate of mercury baths; wet anti-septic dressings are all very useful. Stages of medium severity may

be healed by frequently applying a dry dressing of iodoform, sublimate, creolin or pyoktannin. In very obstinate cases it is sometimes found efficacious to paint the affected part once with chromic acid (10 per cent. water solution), or to apply wet compresses of picric acid (1–2 per cent.). In cases of profuse granulation and induration, or elephantiasis, all remedies fail. A complete recovery to original soundness is then usually impossible, and our efforts must be confined to removal of the excessive granulations and indurations either by extirpation or by caustic remedies.

Straub describes "traumatic" greasy-heels in army horses after exercising over stubble-fields. The clinical appearances are produced by numerous minute superficial skin-wounds together with secondary erysipelatous and phlegmonous inflammation. A similar dermatitis frequently develops during the course of greasy-heels, if the infective matter be conveyed into broken blisters or into cracks and fissures of the skin. Ohlmann observed greasy-heels in several horses after feeding on distillers' waste. The condition was analagous to that met with in cattle. Greasy-heels has been observed as a result of feeding horses upon turf-meal with molasses (Röder, Fröhner). Frank observed eczema on the legs, genitals, back and even head of sucking foals after the mother had been fed on distillers' refuse, and the symptoms were identical with those of greasy-heels in cattle. Kossorotow describes an enzootic occurrence of greasy-heels among the horses of a Russian regiment, due to their pasterns coming into contact with spurge (*euphorbia*). Malzew believes that the streptococcus is the cause of infectious erysipelatous greasy-heels, but his view that every form of greasy-heels is erysipelatous is, however, erroneous.

C.—ECZEMATOUS SKIN DISEASES IN CATTLE.

General Remarks.—Generalised eczema is rarer in cattle than in horses, although nearly the same forms of eruption occur in both species. Both the papulo-vesicular and scaly eczema have been observed in cattle; also an eruption attributed to hunger as well as an impetiginous eczema on the tip of the tail. In cattle, even more than in any other animals, eczema is usually traceable to errors in diet, the so-called "stalk disease," so much discussed in old text-books, being a proof of this. It presents at first a papular eruption, which later becomes pustular, and occurs generally on the extremities.

It is caused by feeding on grape-stalks and vine-leaves. A similar eruption has also been noticed after feeding with maize-bran. The influence of food seems to be much more important in that which, both practically and theoretically, is the most serious of all skin-diseases of cattle, viz., the so-called "malt-eczema," with which the grape-stalk sickness is obviously nearly akin. Compared with this disease, all other non-parasitic forms of eczema in cattle are so unimportant, that it suffices to refer our readers for details of their aspect and treatment to the conditions described in horses. Malt-eczema, on the other hand, demands detailed consideration.

MALT-ECZEMA (*Foot-rash*).

Definition.—Opinions still differ greatly on the nature and definition of so-called malt-eczema of cattle. It is obvious that many diseases, which have etiologically nothing in common, and which as yet can only be clinically distinguished with difficulty, if indeed at all, have been grouped under this title. In veterinary literature, we find that the eruptions produced by feeding on brewers' grains and vintners' waste, on lucerne, white mustard or mouldy fodder, and even mange of the pastern and grease, have been confounded with malt-eczema of cattle.

The occurrence of grease in the ox has been denied by many observers; it is certainly very difficult to distinguish it clinically from malt-eczema, for similar, or even identical, skin-diseases may result in cattle from entirely different causes. The origin of the two diseases is, however, quite distinct, grease in the ox, as in the horse, being due to external conditions such as dirt, neglect, damp or chill, in combination with the influence of micro-organisms; while malt-eczema is directly traceable to feeding with potato-mash. Grease and malt-eczema are, then, two entirely different forms of eczema in cattle. The confusion of terms has led to very diverse views being held as to the nature and origin of malt-eczema.

The various hypotheses regarding the nature of malt-eczema will be discussed later. We hold the view that malt-eczema is a toxic exanthema, analagous to the exanthema produced by drugs, such, for instance, as mercury; that it is induced by a hitherto unknown toxin contained in potato-waste and probably present in the entire plant (solanin ?); and that it usually manifests itself as a vesicular eczema:

Occurrence.—True malt-eczema has only been known for about 80 years. Its first appearance in the twenties of last century is closely connected with the great extension of potato culture and the establishment of large distilleries for treatment of the products, with the consequent increased use of potato-mash for feeding purposes. Spinola first observed the disease in 1827, and described it more fully in 1836. Fattened oxen and male animals appear to suffer most, and after them, fattened cows. Milch-kine, on the contrary, often seem immune. Certain animals have a predisposition to the complaint, while others, though fed on the same food, remain immune. The most serious outbreaks occur in spring and early summer, owing probably to the fact that germination in the potato reaches its climax about this time. Freshly stalled animals suffer most, so that the disease is generally found in byres where the cattle are often changed. Under such conditions cows recently used for milking may, when newly stalled, also suffer. As a rule only the hind legs are affected from the foot to the hock. Sometimes the eruption is confined to the fore-feet, or again, all four extremities may be affected. Other parts of the body, such as the trunk, neck, shoulders, etc., may also be attacked, though this is rare. The condition of the byre has no influence upon the outbreak of the disease. It may occur in the cleanest and best regulated establishments, where straw is good and abundant, and the stall dry.

Etiology and Pathogenesis.—Malt-eczema is most frequently produced by feeding on potato-mash. Other mashes, such as rye and maize, are in this respect innocuous, as are also brewers' grains, although these have been cited by some authors. The intensity of the disease is regulated by the quantity of mash consumed. Exclusive feeding on such mash, or the consumption of large quantities (16 to 18 gallons a day, per head) with but little dry food, is certain to produce it, whereas, if the quantity be more moderate (say 8 gallons), many animals will escape, whilst those which become affected will suffer but slightly. The gravest results arise from germinated potatoes, or from those in a state of advanced fermentation. The singular fact must be mentioned that not every potato-mash is equally injurious. In different years the number of cases in the same byre varies considerably. This may be due to some variation in the fermenting process, *i.e.*, in the products of fermentation, or to certain species or growths

of potato, composition of the soil, kind of manuring, etc. It is possible that the effect of the potato may vary, like that of the lupin, according to the nature of the soil in which the plant has been grown. Baranski states that the disease was first seen in Galicia after the introduction of the Gleason potato. The method of distillation seems to have some influence on the production of the disease, for malt-eczema principally appears after feeding with rapidly distilled mashes. Acidity of the mash does not seem to have any effect on the production of the disease.

Malt-eczema is not exclusively caused by feeding on malts, but may follow consumption of any part of the potato plant. Both raw and boiled potatoes (especially those which are sprouting or germinating), the water in which they have been boiled, and even the potato-tops, not unfrequently produce it. We are therefore forced to infer that the potato-plant itself contains some poisonous principle, which remains behind in the mash, and which, after extraction of the carbo-hydrates from the potato in the form of alcohol, is present in relatively larger proportion. Up to the present nothing certain is known of the nature of this poison. We may perhaps best form an idea of its action by assuming that on its excretion by means of the sebaceous and sweat glands of the skin, it exercises an irritant and inflammatory influence, and thus develops an eczema. This assumption is confirmed by the comparative immunity of milch-cows, in which the toxic agent is removed from the body by the very active secretion of the lacteal glands. The fact, moreover, that the milk of cows suffering from malt-eczema actually contains some poisonous property is proved by its injurious effect on calves and children, as frequently observed, the former being attacked by diarrhœa and the latter by eruptions. In the same way we explain the fact that animals suffer less if actively exercised, and the excretion of this matter be thereby stimulated and expedited. To this theory an objection has been raised in the question, why are, as a rule, only the hind feet affected ? In reply we say that the eruption occurs at times in every part of the body. We must bear in mind that the skin of the hind feet is especially predisposed to affection by the action of wet and dirt from the ground and of the dung, which, after feeding on mashes, is frequently very thin and even diarrhœic. The smaller power of resistance to toxic exanthemata (medicinal exanthemata) possessed by the skin of the extremities we have already noted

in a case of bromine eruption in a horse, which after strong doses of bromide of potassium had an outbreak over the whole body, which was, however, only slight upon the trunk, but intense towards the extremities.

OTHER OPINIONS UPON THE NATURE OF MALT-ECZEMA.

1. The *solanin*, or *solanidin*, contained in potatoes has been condemned by some authors, and malt-eczema considered to be a form of solanin poisoning, an opinion confirmed by the fact that the amount of solanin in potatoes increases at the time of their germination, and that several cases of the disease also occur after feeding on germinating potatoes. In addition to the gastric derangements consequent upon the feeding of cattle with green potato-tops, a vesicular, scurfy eczema frequently appears, upon the legs, in the neighbourhood of the udder, anus and at the root of the tail, and bears a striking resemblance to malt-eczema. It may be described as an exanthematous form of solanin poisoning (irritant action of solanin, similar to that of saponin, when excreted by the skin).

2. The *fusel oil* generated in alcoholic fermentation cannot, for many reasons, be accepted as a cause. It is present in every malt, for instance, in those of maize, barley, and rye, which produce no eruption. Besides, the potato mash is usually given entirely free from fusel oil. Further, there is no possibility of its presence in germinating potatoes or potato tops, and finally, the phenomena of fusel-oil poisoning are known to be quite different from those of malt disease.

3. The *acids* contained in the mash (acetic, lactic, butyric) are found in every other mash, but are absent from potato-tops; moreover, their quantity is so variable that a harmless mash sometimes contains more than an injurious one.

4. Johne's hypothesis, which would bring the high proportion of *potash salts in potatoes* into etiological connection with malt-eczema, cannot explain the production of a vesicular eczema, because the potash-salts act as poison merely upon the muscles, and a large percentage of potash in food stuffs is not injurious.

5. According to Zürn the fermenting *barm-cells* contained in the yeast of alcohol (saccharomyces cervisiæ) change in the bowels to rod-shaped organisms and, in combination with other bacteria and micrococci present in the intestines, produce a mycotic inflammation of the skin when brought into contact therewith through the dung. Johne has very properly raised the following objections to this theory. The same yeast fungi are present in all malts, and therefore every malt should produce this result, which is known not to be the case with malts of rye or maize. The incriminated barm-cells and fungi are found also in the dung of healthy animals fed on potato-mash, and indeed of such as have not been so fed, and must therefore be regarded as merely accidental ingredients of the dung. Then, too, malt-eczema may break out in the cleanest byre, and be absent in the foulest; and parts of the body are often affected which cannot come in contact with the dung. It has also never been shown that sound animals can be infected by the fæces of these suffering from

the disease; attempts at inoculation have always miscarried, and indeed fomentations of warm mash have been employed with success against the disease. Baranski has used this very fact as an argument against Zürn's theory, as a result of his own experiments. Finally, there can be no question of the action of these saccharomyces in cases of malt-eczema following on consumption of raw potatoes, potato-tops, etc.

6. Rabe considers the disease to be symbiotic mange, which *spreads entirely by infection*; that it is connected with the appearance and disappearance of the mite (dermatophagus bovis); and shows an intermittent character corresponding to the manner of life of this acarus. To this Johne rightly objects as follows: The mites are entirely accidental. He frequently found them on healthy animals, as well as after recovery from malt-eczema, whereas he only discovered them on one-third of those that were sick. Furthermore, tail-mange prevails in byres which have always been free from malt-eczema, without any affection of the fetlock or tibia, and without any other animals betraying signs of infection. Moreover, no attempt at inoculation with malt-eczema has ever yet succeeded. The disease can also at any time be cured by merely discontinuing the mash, and that without any further medicinal treatment. Further, tail mange—or rump mange—does not spread with such great rapidity over the whole body. Finally, the connection between malt-eczema and potato-mash is undeniable.

Symptoms.—The local phenomena usually commence about two or three weeks after first feeding on the mash, but earlier when such feeding is exclusive or abundant. There are at first redness and swelling of the skin of the pastern, in consequence of which the animals begin to show stiffness and awkwardness in rising and moving their hind legs. Then vesicles appear upon the skin, which is very painful to the touch, and when these burst they become confluent and form large, moist sores, which dry up later and are often covered with very thick scabs and crusts. The hair is bristly, and the extremities of the affected limbs more or less swollen. This eruption generally extends to the fetlock and may reach even as far as the hock or knee; occasionally in the male it spreads along the inside of the thighs to the testicles, and in the female to the udder. In certain cases, not only the extremities, but the entire trunk, including the belly, breast, neck, back, etc., is covered with impetiginous scales. As a result the swollen skin becomes wrinkled, cracked and split, and discharges a purulent fluid which dries up into scabs. These local changes are accompanied by general disturbance. As a rule the sickness begins with a slight eruption-fever, lessened appetite, retarded evacuation, injection and even increased secretion of the conjunctiva, slavering, etc. Later, persistent diarrhœa often sets in, with rapid loss of strength and flesh, which in extreme

cases, and by no means rarely, leads to complete exhaustion and death. The cause of death then is either exhaustion, septicæmia or pyæmia. The percentage of fatalities is variable. Thus Baranski observed in Galicia a loss of 70 cattle out of 380, or nearly 20 per cent. The severest cases generally occur in dirty, ill-ventilated and badly kept byres, and in older animals, whose powers of resistance are small. Many animals are frequently affected, even from four to six times in a year. Recovery is therefore no guarantee of immunity. The course is, however, as a rule favourable, and recovery occurs in two or three weeks. The scabs gradually fall off, and the hair with them, leaving the skin bare. When neglected, however, the superficial vesicular and weeping eczema may be complicated by severe erysipelatous and phlegmonous, and even gangrenous, inflammation of the skin, which may in turn lead to septic and pyæmic infection (metastases in other organs). Sometimes a peculiar musty, mouldy smell is observed in byres where malt-eczema prevails.

Differential Diagnosis.—Malt-eczema may be confounded with various other diseases, the most important of which are:

1. *Grease.* This may produce an appearance clinically identical with it, but it arises from an entirely different cause. It appears quite independently of feeding on mash, presumably from external causes. The changes produced by grease are also less severe, and we perceive only the erythematous and squamous stages. Yet vesicles also may undoubtedly be produced in grease. In practice we cannot go far wrong if we regard every case of eczema following on a diet of potatoes or potato-mash as being malt-eczema, and all other non-parasitic eczemas of the feet as grease.

2. The *foot-scabies* caused by *dermatophagus mites.* Although we must admit that foot-scabies and malt-eczema may be complicated one with the other, and that the presence of mange-mites on the hind-feet may cause a predisposition to the last-named disease, yet the clinical aspect and course of the two are different, apart from what is shown by the microscope. The eruption in symbiotic mange is decidedly slower and of a milder course than that of the other condition, and rarely attacks the lower extremities without spreading higher up also. Moreover, the character of the resultant eczema is mainly

chronic and squamous, whereas malt-eczema presents chiefly an acute, vesicular or weeping condition.

3. *Foot-and-mouth disease* is also a vesicular exanthema, but it attacks the coronet and the interdigital space, is exceedingly contagious and arises independently of potato-food. Complications of the one with the other are possible, but diagnosis should not be very difficult.

4. *Panaritium.* An infectious inflammation of the terminal phalanges, not rare in cattle, and commencing at the coronet or interdigital cleft, with affection of the tendons, joints, periosteum and bones, can scarcely, as a purely surgical ailment of the hoofs, be confounded with a typical exanthema produced by malt.

5. Finally, the typical character of the eruption in eczema is sometimes so altered by manipulation on the part of the owner, his servants or of empirics (such as rubbing in petroleum or raw carbolic acid) that diagnosis may be somewhat difficult.

Therapeutics.—The simplest remedy consists in immediate discontinuance of potato-mash, or, if this be not possible, in reducing the daily quantity from 16 or 18 gallons to 4 or 8, and adding thereto a corresponding amount of dry or other food-stuff. Märker says that an addition of maize to the potato-mash will prevent malt-eczema, so long as not more than 15 to 17 gallons of food be served daily, about one-third of which should be maize. Abundant exercise of the sick animal may also procure some relief. A great number of medicaments are in use. Rabe recommends a liniment of tar and soap (one part tar, two parts soap, 1 part flowers of sulphur, two parts alcohol), or 5 per cent. carbolic oil. Others use desiccant and astringent remedies in solution or in solid form, *e.g.*, powdered oak-bark, decoctions of the same, lead-lotion, blue-stone, sugar of lead. We might also recommend the application of creolin or lysol in the form of a liniment (creolin and green soap of each 3 oz.; alcohol, 1½ oz. to 15 oz.); or of the ointment (5 to 10 per cent.) or of lysol or creolin, in a 2 per cent. watery solution. Of creolin spirits (4 to 10 per cent. solution). The greatest cleanliness in the byres is essential. Although the origin of the disease is not traceable to dirt, yet an excess of it may always produce a predisposition thereto, and aggravate otherwise slight cases.

D.—ECZEMATOUS SKIN DISEASES IN SHEEP.

General Remarks.—Non-parasitic skin affections in sheep are as yet little known and somewhat rare. According to Haubner-Siedamgrotzsky, a kind of rash, in reality a papular eczema, is occasionally met with which betrays its presence by tufting of the wool. A squamous eczema is also observed in the course of distomatosis and other cachectic affections, in which superficial layers of the epidermis scale off. In other instances this condition is traceable to neglect and bad attendance, and is called "dirt-mange." May also speaks of a pruritic vesicular eruption as well as of impetiginous eczema under the names of "Prurigo" and "scabby-mange." Greasy legs likewise occurs among sheep, although the genuine malt-eczema has not yet been observed. Pustular eruptions appear about the mouth after eating mouldy fodder. A condition which frequently occurs among sheep and which must not be confused with eczema, is seborrhœa, *i.e.*, excessive secretion by the sebaceous glands.

The commonest and most important non-parasitic eczema among sheep is the so-called "rain-rot" (moist or "fatty" mange). In badly-nourished animals with open fleece the superficial layers of the epidermis become macerated during continuous rain or damp weather, and a weeping eczema develops under it, with swelling of the skin, formation of vesicles and scabs, loss of wool, itching, etc. It attacks especially the back and loins, but may also spread forwards upon the shoulders, neck and head, and backwards to the root of the tail. Recovery usually follows spontaneously on cessation of the rainy weather, so that treatment is seldom necessary; but in severe cases the sheep must be brought under cover. Injury is generally restricted to falling-out of the wool. The condition may be mistaken for mange; for differential diagnosis c.p. malt-eczema.

E.—ECZEMATOUS SKIN DISEASES OF PIGS.

Occurrence.—Among non-parasitic eruptions in pigs the so-called "Rust" of young pigs ("pitchy-mange," "scale-eruption") is perhaps the most important. This presents at first a vesicular and later a pustular and scurfy eczema, which is most frequent in young and weakly or sickly animals, generally

spreads over the whole body and may arise from a variety of causes (dirty styes, accumulation and decomposition of the sebaceous matter of the skin, etc.). We usually first find vesicles, which quickly change into pustules and then dry up into thick, black, pitch-like scabs, beneath which a serous exudation takes place. In most cases this eruption is but a secondary symptom of some internal constitutional ailment, and may be compared to the scrofula of man. Treatment consists in cleanliness, frequent washings with soap, and good food. As the sarcoptic mange of pigs produces similar changes on the skin, it is also sometimes termed "rust." But it differs essentially in being contagious. Microscopic examination will guard against error in diagnosis.

Granular Eruption.—Under this name Zschokke describes a peculiar chronic vesicular eruption in pigs, which occurs in patches the size of a hand on the ears, back, loins and root of the tail, and is marked by the greyish-violet colour of its blisters, which are very like the colour of small shot. The vesicles never burst and cause no itching (differing from rust). The process, says Zschokke, consists in a cone-shaped proliferation of the epidermis inwards, induced by specific micrococci. Ostertag and Johne regard this granular eruption as multiple dermoid cysts; while Lungershausen looks upon it as an arrested development (hypotrichosis). According to Olt, the ailment should be considered as a coccidiosis of the sweat glands, whereby the coccidia, after immigrating into the epithelium, provoke the formation of retention-cysts. Voarin has succeeded in conveying the disease from sick to healthy swine by contact. Lühe doubts the relationship of the "coccidium fuscum" to the coccidia. In his opinion the granular eruption of swine is in need of fuller investigation.

Pityriasis Rosea.—Schindelka thus names a skin-disease of pigs in which red and slightly raised spots about the size of a lentil appear on the breast and belly in somewhat circular form. These spots, at first round, have a diameter of ¼ inch and seem to be made up entirely of small nodules. Later they grow flatter and paler in the middle, spread outwards, run into each other and form sinuous lines. Duration, three weeks. Schindelka has observed the condition only in quite young pigs and in the spring. Its occurrence in all cases was spontaneous and in no instance was it conveyed to other animals. The disease usually terminated in recovery.

URTICARIA, NETTLE-RASH, NETTLE ERUPTION, POMPHOUS ERUPTION.

General Remarks on Definition and Causes.—Urticaria is manifested by sharply defined, flat, raised swellings of the skin, the result of a quickly occurring serous transudation in the papillary bodies and the corium. It may therefore be

regaided as a circumscribed œdema of the skin, or an acute localised serous dermatitis, which has, however, only reached the point of exudation of serum and not of infiltration with cells. The lesion of urticaria is clearly distinguished by this from a papule, and with this fact is connected also the possibility of the rapid disappearance of the rash by resorption of the serous fluid. The cause of this transudation of fluid is to be sought in a sudden dilatation of the capillaries in question, consequent on abnormal excitement of the peripheral vaso-motor nerves, or in an embolism. Urticaria may accordingly be looked upon as a vaso-motor neurosis or angio-neurosis, with abnormal permeability of the capillary walls. This affection of the vaso-motor nervous system may be due to skin irritation or may be of internal origin.

1. Among the *external causes* we may notice the action of irritant substances upon the skin, such as insect-bites, stinging nettles (urtica urens and dioica) and the hairs of caterpillars (nethocampa processionea). Other irritants may produce a nettle-rash on tender and predisposed skins, such as rubbing turpentine on a horse with colic.

2. *Internal causes* can only produce urticaria by disturbances in the innervation of the vaso-motor skin-nerves and only in animals which are predisposed. This predisposition is probably some disturbance of the vaso-motor nerve-system. Young, full-blooded and well-fed animals are especially subject to nettle-rash. The internal causes may also be presumed to be some irritant taken into the blood which, granted the necessary predisposition, has a peculiar effect upon the skin. This seems to agree with the occurrence of urticaria during the course of several infectious diseases (petechial fever, strangles and the nettle-fever of swine), in which case the virus is responsible for the production of the inflammation. The exanthema of nettle-rash produced by certain disturbances in the intestinal canal (gastro and intestinal catarrh, icterus) must also be regarded in the same light, certain abnormal products of decomposition and digestion (toxines) are absorbed by the blood, and excreted by the skin, producing inflammation. As in man, so too in animals, there is a certain idiosyncrasy or individual susceptibility to particular external influences, and especially to the action of certain kinds of food, which may be supposed to cause urticaria. Thus pod-fruits, buck-wheat, green potatoes, green oats or rapid changes in fodder are all blamed. In man, strawberries, raspberries, crabs, oysters and salt-water fish

produce the eruption on persons who are predisposed. In cattle, urticaria appears to be induced reflexively by conditions of mental excitement. In the cases described by Tapken, the clinical history generally shows that urticaria developed in the cow either during the preparation of food or after fighting with another cow. Schindelka has seen it repeatedly occur in the dog in response to an excitement of the sexual organs. Finally, nettle-rash may occur in the course of other complaints. Thus Schindelka observed it during the so-called Canadian or English horse-pox.

3. *Rapid cooling of the skin when the body has been overheated* seems also to be an exciting cause of the eruption. It consequently occurs very often in spring and summer during great heat, in heavy torrential rain, after exhausting labour or rapid movement, as well as sometimes during rheumatic hæmoglobinæmia. In all such cases it may be caused by retention or generation in the body of injurious matter as a result of chill, rather than by any direct influence of the same upon the skin.

4. Nettle-fever has been observed in horses as a specific infectious disease, 157 horses in one battery having suffered from it within a short time.

5. Finally, it is not possible in many cases to attribute the occurrence of urticaria to any specific cause.

Occurrence.—From the foregoing it is evident that urticaria represents in by far the majority of cases a purely symptomatic exanthema, and ought, therefore, to be cited only as an accompanying symptom of the particular disease with which it is associated. As nettle-rash is often the only visible phenomenon, and the internal complaint cannot in all cases be surely demonstrated, we have decided to follow the usual practice and discuss the eruption here.

The ailment is most common in horses, dogs and pigs, and after them, cattle. Among 70,000 dogs taken to the Berlin dog-hospital during nine years, there were 108 cases, or 1.5 per thousand, of urticaria. On practical grounds it is advisable to treat the complaint in horses, dogs and cattle together.

I.—URTICARIA IN HORSES, DOGS AND CATTLE.

Symptoms.—The urticaria of horses, dogs and cattle, formerly known as "heat-boils," or "boil-fever," is characterised by an extremely rapid appearance of pomphi, which in the

course of a few hours or overnight will spread over a large area. At first there are isolated patches of soft swellings the size of a pea, above which the hair looks rough, and which appear mostly upon both sides of the neck, round the shoulders, along the back, sides and buttocks. These multiply and grow quickly, very often coalesce and then form patches as large as the hand or a dinner-plate, when even the head, fore-legs, lower thighs and udder may be attacked, giving the animal a very unsightly appearance. Pruritis is only occasionally present. Pug-dogs seem to be most readily affected.

Sometimes the pomphi are also met with upon the visible mucous membranes, as of the nose, mouth and vagina. Their presence in the nose may lead to disturbance of breathing (Schleg). Zipperlen found the conjunctiva in a cow very much swollen, and the mucous membranes of the rectum and vagina pressed out in thick rolls. If the transudation penetrate between the rete Malpighi and the epidermis, vesicles may develop upon the pomphi (so-called nettle-blisters, pomphosis); which usually produce intense itching and loss of hair. In addition to these processes upon the skin and mucous membranes, various disturbances of the general health are perceived, according to the nature of the chief causal malady. Very often symptoms of slight fever—seldom of high fever—are an accompaniment. The differentiation between nettle-fever and simple feverless nettle-rash, formerly based on this fact, is consequently of no avail. Weariness, depression and disinclination for work are associated with the condition, or signs are observed of gastro-intestinal catarrh, loss of appetite, diarrhœa, constipation or slight jaundice. In the case of a horse, temporary outbreaks of sweating were noticed with the urticaria. Usually the eruption vanishes as quickly as it appears, say in one or two days. Fresh attacks often recur, which make the course longer and even chronic. Some animals indeed suffer several times in one year from urticaria.

Therapeutics.—Treatment is generally superfluous; in many cases fasting and warm covering suffice. If there is evidence of gastric disturbance, attention to the bowels may be advisable; give purgatives, such as tartar emetic, aloes, eserine, arecoline, calomel, or, as a milder evacuant, Epsom or Glauber's salts. In rare cases, where dyspnœa is present to a dangerous degree, tracheotomy may be necessary (Albrecht).

2. URTICARIA IN PIGS (*Nettle-fever, Brick-rash*).

Definition.—The nettle-fever of swine, "Brick-rash" (Danish nodular erysipelas), was in earlier decades regarded as a milder form of erysipelas. Following Haubner's example, it has, on practical grounds, been separated therefrom and treated as an independent disease. In later years nettle-fever has again been indicated as a complaint allied to the erysipelas of swine, according to the bacteriological investigations of Jensen, Lorenz and others. They show that the erysipelas of pigs runs sometimes benignly as nettle-fever, and sometimes malignantly as true swine erysipelas, again as a necrotic inflammation of the skin, or furthermore as a chronic endocarditis, etc.

If these statements are confirmed, we must assume that, like other infective diseases, swine erysipelas may run a benign course as an exanthema possessing the aspect of urticaria; and that many cases of so-called nettle-fever must be regarded as erysipelas. It is certain that, as in other domestic animals, so also in the pig, cases of urticaria occur from unknown causes, such as gastric disturbance, and are quite independent of the disease occasioned by bacilli. There is, therefore, no conclusive reason why we should omit the consideration of nettle-fever from this chapter. Schindelka, the greatest authority upon the skin diseases of animals, does not support the view that all cases of urticaria in swine are to be regarded as erysipelatous. We differentiate between:

1. An *independent, non-infectious* urticaria; and
2. A *symptomatic* urticaria, which is presumably produced by *bacillus-erysipelas* (comp. chapter on Swine Erysipelas, Vol. II.).

Symptoms.—The phenomena of non-infective urticaria (which usually appears sporadically) consist in a rapidly appearing eruption of pomphi on the skin, which vanishes just as quickly and is accompanied by gastric and feverish disturbances. On animals previously sound there appear, and usually overnight, a number of pomphi, about the size of a hazel nut, which are painful when touched, rise $\frac{1}{25}$ to $\frac{1}{12}$ inch above the surface, and are about the area of a crown-piece, but by the confluence of several may become as large as the palm of

the hand. They appear mostly on the upper trunk, breast, loins, upper thighs and belly. These pomphi vary in colour according to the stage and intensity of the condition. At first they are merely red spots, rising a little above the surface, whereas in the advanced stage they are white elevations with a red margin (so-called white nettle). Sometimes a serous exudation is also to be seen on the top of the pomphi, which dries up to a scab and is cast off. The general symptoms are lessened appetite, as well as an invariable constipation and at times also vomiting. The animals seem melancholy and dull, bury themselves in their straw, are disinclined to rise, show strongly injected mucous membranes, accelerated breathing and medium fever (about 104° Fahr.). In a few cases the gait is stiff and awkward, which points to some rheumatic affection of the muscles.

Prognosis.—In contrast to erysipelatous urticaria, nettle-rash (which is usually not infective) follows an exceedingly benign course. In slighter cases recovery takes place in one or two days, in severer ones, after four to six at most. No fatal case has yet been known. An improvement is indicated by abatement of the constipation, when the exanthema vanishes, the pomphi grow paler, become soft and withered and then quickly disappear, which ends the attack. In the few cases ending fatally the condition was in all probability an unrecognised erysipelas.

Treatment.—Owing to their benign aspect and typical course, ordinary cases of nettle-fever require no treatment, or merely dietetic. To relieve the constipation we should administer clysters, soap-enemas, etc., according to symptoms, and also give doses—not too small—of calomel (15–60 grains). Haubner recommends saltpetre (1½ dram) with glauber salts (1½ oz.); with honey for licking or as electuary. We may also reduce the inflammation of the skin by frequent douches of cold water.

GANGRENOUS DERMATITIS.

(Gangrene of the white marks.)

Etiology.—The causes of gangrenous dermatitis in horses and cattle, which specially attacks piebalds and animals with white markings, and is occasionally enzoötic in its spread,

must be sought exclusively in the action of external irritants. As a result of intensified erythematous and eczematous inflammation, the affected portions of skin ultimately become gangrenous. These irritants are of different kinds. 1. The *intense power of the sun*, or of a hot, dry wind, may in many cases be regarded as the direct cause of this condition (so-called sun-gangrene, gangræna solare). This represents a higher degree of erythema solare (thermal activity of the red rays, giving rise to inflammation). According to Plassio it is noticed in Africa in horses with unpigmented skin upon the parts unprotected by saddle or harness. 2. The fact that the skin is *unpigmented* causes a *pre-disposition* not only to the action of the sun, but also to that of other irritants, as lacking the protection of the pigment. Consequently the white marks on the head and extremities and the light unpigmented skin of parti-coloured horses are most readily affected. 3. Certain *fungi*, it is evident, are also able to produce a gangrenous dermatitis in many cases, viz., in the so-called clover-sickness, and perhaps also in lupinosis, and this occurs especially on the white parts of the skin. Spoilt vetch and green food covered with mildew (erysibe fungi), as well as plant-lice, are said to cause this inflammation. 4. The so-called gangrenous or discharging greasy heels are due either to an increase of the irritation usually present in greasy heels (severe frost, etc.), or to some infective process. Greasy heels sometimes appears enzoötically among horses. It may also occur as an enzoötic eczema, distributed over the whole of the body.

With regard to the dry, diffuse gangrene of the skin in pigs, see the chapter on Swine Erysipelas (Vol. II.); and for mummification of the extremities see that on Poisoning by Ergot of Rye (p. 227).

Symptoms.—Gangrenous inflammation of the skin begins with the appearance of erythema or eczema. The swelling of the skin rapidly increases, cracks and fissures are formed and necrosis of certain patches quickly sets in, which is at times only superficial and at others very deep. In the former case leathery scabs appear upon the skin; and in the latter the whole skin shrivels up like parchment right down to the subcutis, and is all thrown off by suppuration. This casting-off of the necrotic parts takes place with formation of a line of demarcation. Abscesses and fistulæ may also be developed. Healing is produced by granulation and formation of scabs.

Therapeutics.—Treatment consists at the outset in rubbing in lead or lead and tannin ointment, carbolic oil, carbolic ointment, iodoform salve or creolin ointment, creolin-liniment, creolin-water, and in the application of pyoktanin, etc. Later, *i.e.*, after necrosis of the skin, the necrotic portions should be removed by knife or scissors and an antiseptic dressing applied. The feet should be bathed with warm antiseptics.

BULLOUS DERMATITIS, VESICULAR ERUPTION, PEMPHIGUS.

Definition.—In bullous dermatitis we have a formation of blisters, as large sometimes as a hen's egg or the palm of the hand, rising upon an intact or reddened skin, whereas in eczema small blisters only occur. Nothing positive is known as to the cause of this singular exanthema in our domestic animals.

Symptoms.—Various reports on the phenomena have been published. As a rule the course of the disease is acute (pemphigus acutus). Loiset saw the eruption appear enzoötically in cattle. Large, round or oval blisters, as much as four inches in circumference, showed upon the loins, croup and thighs, charged with a clear fluid and, soon bursting, left behind them wet places, which scabbed over and then healed with desquamation of the skin, leaving shiny patches. Seaman noticed a similar eruption in an ox, accompanied by feverish shiverings, in which the blisters were the size of a hen's egg. Lucet discovered the streptococcus pyogenes in the blisters on a cow. Dieckerhoff observed the eruption on five horses. On the belly, head, neck, thorax and between the thighs, blisters, from a walnut to the palm of the hand in size, arose upon the skin, full of a fluid, either clear or of a yellowish tinge. Usually the uplifted epidermis broke, leaving large sores, from which oozed a fluid secretion. Apart from the violent itching, there was no disturbance of the general condition. Similar cases in the horse are reported by Barnick, Gutzeit, Wessendorf, Sauer, and Zinnecker. Graffunder describes a case in a horse which lasted about 12 days, and in which blisters formed on the skin, while dark red spots the size of a pea appeared upon the nasal mucous membranes. Doges saw one case in a mule and Winkler and Gutbrod have observed it in swine.

Pemphigus.—This bullous dermatitis has a great resemblance to the vesicular eruption in man known as pem-

phigus, the etiology of which is also still very obscure. The latter, as seen in man, occurs sometimes as an acute infective disease, and then a vaso-motor neurosis, and again a vegetable parasitic disease of the skin. The above-mentioned cases in cattle seem to be especially like the pemphigus vulgaris of man, which also runs a benign course; whereas pemphigus foliaceus finally leads to death. Fröhner describes a case of chronic pemphigus in a dog, which terminated fatally; there were numerous water-blisters and ulcers over the whole of the body, with extreme emaciation—the condition lasting over many weeks. Schindelka (Hautkrankheiten, 1903, p. 262) writes of true pemphigus in the domestic animals as follows: "Pemphigus appears to be extremely rare among the domestic animals. There is a large amount of clinical material at my disposal, but I have never yet met with a single case. The greater number described as pemphigus in veterinary literature, do not, upon closer investigation, correspond with the clinical picture of that condition. Among the domestic animals, one case only has been reported which presents the symptoms and runs the course characteristic of true pemphigus. This is the case of a dog, described by Fröhner."

Herpes labialis.—In addition to this bullous dermatitis characterised by large blisters, some authors have described a specific vesicular eruption (especially in horses) upon the lips and nose, which they call "herpes labialis." The exanthema is characterised by small, quickly developed blisters, grouped together in clusters, which attain the size of a lentil, contain a clear fluid, and soon heal with formation of scab. It is observed upon the lips of horses which have been out at grass, and occurs also in glanders and influenza and as an accompaniment of gastric disturbances (Röll, Siedamgrotzky, Schindelka). Peter and Schossleitner have observed the condition—which is quite unmistakable—as an enzoötic among both old and young sheep. It must be admitted that this specific eruption much resembles the herpes facialis and genitalis of man, and so far agrees with it that it occurs in the neighbourhood of the natural orifices of the body. But so much misuse has been made of the term "herpes" in animal pathology that it would certainly be more appropriate, following Gerlach's suggestion, to restrict its use to a specific parasitic skin disease, viz., herpes tonsurans. With the herpes zoster (girdle-erysipelas) of man which appears in the course of a nervous complaint, the benign exanthema described above as a specific vesicular eruption has certainly nothing in common.

Impetigo.—This term includes all eruptions characterised by large pus blisters (the exanthema which occurs in dogs after suppurating mange; Canadian horse-pox; swine-fever; cattle-fever). Schindelka observed an impetiginous exanthema upon several occasions in old bitches. It was not caused by mange and appeared in the form of pustules as large as hemp-seeds upon the teats and the inside of the thighs.

LOSS OF HAIR AND WOOL (*Alopecia*).

Occurrence.—The falling out of hair and wool occurs as an independent skin affection, apart from other skin diseases, sometimes locally and sometimes over the whole body, being known in the former case as area celsi. The nature of alopecia must be sought in certain disturbances in the nutrition of the skin (tropho-neurosis) which cause atrophy of the hair-roots. The co-operation of parasites cannot be absolutely disproved, though up to the present not clearly established.

Etiology.—The causes of alopecia are partly known. A general falling out of the hair presents obviously a symptom of some general disturbance of nutrition and is therefore termed alopecia symptomatica. It is noted where the food conditions are bad; as a result of scanty feeding, and on the contrary in great obesity; during pregnancy; during suckling; in the course of prolonged consumptive disease; during certain blood disorders, such as petechial fever; after protracted sweating; after certain interruptions in the circulation of the skin; as an inherited and congenital ailment (alopecia congenita); after eating certain kinds of food, such as sour hay from swampy soil; or after psychical excitement. The area celsi (alopecia areata) is regarded by some as a tropho-neurosis, that is, a disturbance of nutrition in the region of the skin-nerves, and by others as a parasitic affection.

Symptoms.—1. In *alopecia symptomatica* a general dropping out of hair occurs all over the body, after which the animals appear perfectly naked. In horses the alopecia is sometimes preceded by gastric disturbance, and also by œdematous swellings on the limbs, under the belly and breast. In other cases no accompanying signs can be perceived. As a rule, the hair grows again in a few weeks. From our observations alopecia in dogs is very common, especially upon the back. In Berlin 224 cases (3 per 1,000) were observed among 70,000 cases treated in the dog-hospital in nine years. A darker pigmentation of the bare surfaces is usually noticed as an accompaniment, which probably acts as a protective substitute for the hair. Siedamgrotzky found on examining such pigmented spaces under the microscope that, in addition to atrophy of the hair and hair-bulb, the cells of the rete Malpighi were

quite infiltrated with black and brown granules; the same also in the root-sheaths of the hair-follicles and the gland-cells of the sebaceous glands. From sheep the wool usually falls out in coherent masses, leaving large naked patches.

2. *Alopecia areata.*—This shows itself at first in small, round, bald spots, which gradually grow larger. It is commonest in dogs and resembles very much the appearance of herpes tonsurans, only that the fungus characteristic of the latter cannot be found. Röll noted a case of a horse in which the hair came off at first only on very small spots, but wherein the alopecia spread in the course of a year over almost the entire skin. Here also abundant black pigmentation was present with atrophy of the hair-bulbs. No micro-organisms could be found, so that some atrophic process in the skin must be assumed as the cause. The complaint is very obstinate and defies all treatment.

Therapeutics.—There are several stimulating remedies which may be tried to promote regrowth of the hair in alopecia. Spirit is used most frequently, or spirit of soap and camphor. Also tincture of cantharides, if sufficiently diluted, may be rubbed on (1 : 5 spirit, or with fat as a pomade). In alopecia areata we recommend, in addition to the anti-parasitic Peruvian balsam (1 : 10 spirit), creolin (1 : 10–20 spirit) and tincture of iodine mixed with equal quantity of spirits of wine and laid on with a brush for several days successively.

Trichorrhexis nodosa.—A peculiar affection of the hair, evidently identical with that described by Kaposi under this name in man, has been observed by Trofimo. Among nearly two-thirds of the horses in an artillery brigade symmetrical spots appeared on the back, loins and croup, on which the hairs were all broken off about a third of an inch from their root, or swollen out into a nodule. The place of fracture was found under the microscope to be considerably fibrillated. A similar interesting case is described by Mégnin. Römer observed the condition in the hair of the hind fetlocks. This hair-affection seems to be common in the Prussian army, and in one year was found in eight regiments. According to Tennert, it is more common among army horses than is generally supposed, for he has noticed it in every regiment to which he has been attached. Steinhardt's description is that small, greyish-white nodules appear upon the diseased hair at distances of from $\frac{1}{8}$ to $\frac{1}{6}$ inch and progressing from the tip of the hair to the root. At these nodules the hair bends or breaks off. Both the protective hair (mane and tail) and the covering hair are affected. Upon the latter, irregular patches of such broken hair are found about the size of a crown piece. Treatment with sublimate, creolin, carbolic acid, tar, ichthyol, etc., is fruitless, and the hair is not

renewed until the change of coat. According to Kalkoff and Seegert, a daily washing of the affected places with a five per cent. watery solution of pyrogallic acid was found effective. Kutzner and Reichert recommend painting with a 1 per cent. solution of pyoctanine, the part being previously washed with soda and water. Tennert does not consider that any remedy—not even pyrogallic acid—can do much to benefit the condition. Faletti finds a 2–5 per cent. solution of perchloride of mercury most efficacious. It is thought that the complaint is transferred to other horses by the grooming utensils (also in the beards of the attendants). The causes of trichorrhexis nodosa have not been definitely ascertained. (Abnormal dryness with splitting of the hair? A specific vegetable micro-organism?)

HÆMATIDROSIS. BLEEDING OF THE SKIN (*so-called Bloody Sweat*).

Etiology.—The numerous cases of bleeding of the skin described in veterinary literature under the name of "bloody sweat" have a very obscure etiology. Many of them are probably purely symptomatic hæmorrhages occurring in the course of severe general disease, such as petechial fever, anthrax, septicæmia or scurvy. Others belong to the realm of acute exanthemata, which may sometimes be complicated with hæmorrhage. If we omit these, there still remain a number of peculiar cases of bleeding of the skin, which have not arisen through secretion by the sweat-glands, as their false designation, "bloody sweat," might lead us to suppose, but represent hæmorrhages either by rhexins or by diapedesis. A definite cause for this cannot be discovered, but probably a general disturbance of nutrition of the vessels lies at the base of it. To this class belong especially the skin-hæmorrhages of cattle. Besides these hæmorrhages there are certain bleedings of the skin which occur particularly among thoroughbreds and horses of oriental, Hungarian, Russian and Tartar breed, which are harmless in their nature and were formerly considered to be nature's self-aids. Probably these latter arise from the settlement of nematodes in the skin (filaria hæmorrhagica) and from the vascular injuries thereby effected (hæmatidrosis parasitaria). They are not infrequent in horses imported into Germany.

Symptoms.—The extravasation of blood occurs in drops and in various parts of the body. In slighter and benign cases it affects the skin only, usually on the shoulders, neck, breast and extremities. In those cases the bleeding is slight and

soon abates. If, however, a general hæmorrhagic diathesis be present, we have hæmorrhage into the mucous membrane, and bleeding occurs from the nose and intestines, when we see bloody milk and bloody urine. From this severe anæmia often develops, of which the animal sometimes dies. General disturbances such as slight fever, gastric disorder, etc. also usually accompany this last kind of hæmorrhage. In horses of pure blood the bleeding occurs sometimes in a stream for a brief period.

Therapeutics.—A treatment of partial skin-hæmorrhage in thoroughbreds is seldom necessary; at most we may wash the bleeding places with cold water, or try anti-parasitic remedies. But for general hæmorrhagic diathesis an internal styptic treatment must be adopted, viz., administration of ergot, tannin, sugar of lead, etc.

ACNE AND FURUNCULOSIS.

Definition and Causes.—Under the name of acne we understand a suppurative inflammation of the follicles of the skin (suppurative folliculitis), which has arisen in consequence of accumulation and decomposition of the matter secreted by the sebaceous glands. The provocation to such inflammation may come either from without, or from the blood and penetrate to the sebaceous glands and follicles. The skin-affection is local but deep, and reveals itself either in papules or pustules. A typical form of acne arises from incursions of the acarus folliculorum into the follicles (a pustular form of acarus eruption). In contrast to this parasitic acne, which will be discussed elsewhere, the common acne is a non-parasitic or non-infective skin disease, which is oftenest seen in dogs on the head where the muzzle fits, as a consequence of persistent irritation. Acne also occurs in horses, usually under the harness (neck, shoulders, back), in cows upon the udder, and also in Negretti sheep on the inner surfaces of thighs and abdomen. Schindelka often noticed a so-called tar acne (acne artificialis) after frequent rubbing of horses, dogs and swine with wood-tar.

By the name of furuncle we describe large acne pustules which cause necrosis of the follicles (gangrenous, suppurative folliculitis). It is therefore a development of acne and represents only a higher degree of follicular inflammation.

Comedones, or acne punctata, is the name given to retentions in the sweat follicles. The condition occurs in dogs and swine, and is unimportant.

Symptoms.—1. The acne of *horses* has been described under many names, such as nodular, tubercular, pimply rash, knotty scabies, heat-nodules, tallow-nodules, pustular measles, callous tubercles. Many of these skin diseases do not, however, belong to true acne, but to papulo-vesicular eczema, from which it is not always easy clinically to distinguish. It is most frequently found under the seat of the saddle as small, firm and painful nodules, from which little tallowy or pus-like plugs may be squeezed, and on the summit a yellow-brown exudation or dry scab may often be found. It arises after severe exertion from the prolonged irritation of the saddle and consequent decomposition of the skin-secretions, with subsequent infection of the sebaceous glands. Similar nodules, though generally somewhat harder and larger, up to the size of a pea, are often found beneath the harness. After the tallow or pus-plug is pressed out, cup-shaped ulcers remain and with very narrow entrances and jagged edges. In general the acne-nodules on horses are distinguished by the fact that their contents consist less of pus than of a tallowy, greasy matter like groats. The surrounding skin sometimes shows signs of chronic indurating dermatitis (sclerosis), as well as of eczema squamosum. Contrary to papulo-vesicular eczema, we seldom find acne spread over the whole body, but rather confined to certain predisposed parts, such as those under the harness and saddle.

2. In *dogs* the disease is generally localised upon the skin at the back of the nose. It usually occurs in bull-dogs, mastiffs, pointers and Leonberg dogs, that is, in those with a long forehead. The pressure of the muzzle causes at first a superficial inflammation, which spreads to the more deeply seated follicles, causing suppuration with a swelling-up of their orifices. The skin feels thick and hard and, on pressure, discharges at various points a bloody or purulent matter. Nodules the size of a lentil, pea or even a hazel-nut are also present, with a sunken purulent centre. In addition to the back of the nose, we find acne-nodules and pustules, or the furuncles produced thereby, upon the lips, forehead and temples. In rarer cases furunculosis occurs upon the skin of the trunk and extremities, in patches as large as a five-shilling piece or the palm of the hand, which

are interspersed with nodules and pustules from which a bloody-purulent secretion can be squeezed. Sometimes the pustules run together into large abscesses, above which the skin has a bluish red or violet colour. This form of furunculosis we find mostly in griffons and long-haired pointers. Its causes are unknown, and are perhaps to be sought in a congenital difference of structure in the sebaceous glands. When found on the trunk or extremities the prognosis is very unfavourable, as it always spreads and may take months to heal. The complaint is much more frequent among the dogs of large towns, owing to the muzzling orders often in force there. Of 70,000 treated in the Berlin dog-hospital in nine years, 462 (or 0.7 per cent.) suffered from a high degree of acne and furunculosis.

Therapeutics.—The treatment of acne and furunculosis is mainly surgical, consisting in pressing out, opening and cleaning the abscess-foci. Then disinfectants must be introduced into the open follicles, for which we have found an ointment made of Peruvian balsam and creolin (1 : 10) especially useful on horses and dogs. Cauterization of the hollows with a nitrate-of-silver pencil is also very advisable. Frick recommends burning with Paquelin's cautery. In very obstinate cases, Schindelka advises the employment of keratolytics in the form of pastes (salicylate ; soft soap with sulphur ; glycerine with iodine). A radical cure, especially in dogs, is sometimes only to be obtained by total extirpation of the affected portion of skin. As a prophylactic we recommend avoidance of all external irritants. In furunculosis of the ridge of the nose the muzzle must be kept off for a time, or at least lined with soft felt, to prevent relapse. Slighter attacks of acne in horses heal of themselves on removal of their exciting cause. Schindelka has seen obstinate acne in dogs overcome by ichthyol and arsenic administered by the mouth.

The remaining anomalies of the skin belong partly to the department of general pathology, such, *e.g.*, as excessive sweating (hyperidrosis), which may be either general or local, upon one side or upon both ; extreme secretion by the sebaceous glands (seborrhœa), which occurs in the horse as seborrhœa sicca (a form of scurfy tetter), and in the dog and in sheep as s. sicca and s. oleosa (grease-scurf), the so-called hardness of skin, sclerodermia of swine, anomalies in the change of coat and formation of pigment ; or else they are purely of surgical character, such as warts, horny skin, elephantiasis, ichthyosis, phlegmona, erysipelas, emphysema, etc.

II.—VEGETABLE-PARASITIC SKIN DISEASES (*Dermato-mycoses*).

General Remarks.—Of skin diseases caused by vegetable parasites on our domestic animals only three have so far been positively demonstrated: 1. Ringworm (tinea tonsurans); 2. Honey-comb scab (favus); and 3. Dermatitis pustulosa contagiosa canadensis (the so-called English horse-pox). Of these the two first are by far the most important. The question has often been raised whether they are caused by two distinct fungi or by two variations of one and the same fungus. The results of microscopical investigation indicate a unity of fungus species, as no essential difference can be found between the hyphæ and conidiæ in tinea tonsurans and favus. But the later studies of Grawitz, who at first accepted the identity of the two fungi with each other, and also with the oidium albicans, have now proved that tinea tonsurans and favus are produced by two separate species of fungus. It is true the spores of both are very similar, but they show many differences in growth, liquefaction of gelatine, form of cultivation and conditions of fructification. Thus in the microscopic aspect of favus the mycelium predominates, while in tonsurans the spores are most numerous. Oidium lactis has nothing in common with them. According to Quincke, favus alone may be produced by three distinct fungi. A further argument in favour of their being two different dermato-mycoses lies in the fact that the clinical aspect of both is quite different, and that intermediate forms are usually absent. On this latter ground alone we must rank tinea tonsurans and favus as two essentially distinct skin affections.

Besides the three dermato-mycoses just named, other skin diseases have been described in which fungi have been found which could not be more nearly defined. Thus Siedamgrotzky reported one clinically much like herpes circinnatus, in which, however, the presence of trichophyton could not be proved. Similar observations we have ourselves often made with dogs. Leisering describes an eruption producing baldness round about the orifice of the cloaca in a cock, which was transmitted by copulation to the hens, upon whom greenish-yellow conidiæ were found. He and Engel have also reported on several vegetable-parasitic affections of the root of the tail and its hair in horses.

TINEA TONSURANS (*Bald Scab. Ringworm*).

Etiology.—The various diseases known as ring-worm, depillatory dry scab, scab-mange (dermato-mycosis tonsurans,

tinea or porrigo decalvans, tinea scutellata), and in calves as dough-mouth or doughy evil, doughy mange and mouth-mange, in other animals as lamb-mange and goat-mange, only merit their title of " mange " in so far that they are all caused by a vegetable parasite. This fungus, which is called trichophyton tonsurans, belongs to the hyphomycetes and was discovered by Malmsten in 1845. As the cause of ringworm in our domestic animals it was first identified by Gerlach, who in 1857 published a classical work on the mange of cattle. The transfer of the fungus to other animals occurs through the combing and brushing apparatus, harness, covers, or by contact with neighbours or, in sucklings, during sucking. Bulls in particular may transmit the affection to a whole herd. During pasturage it may also be easily spread, as then the animals come much in contact.

Cattle are most readily affected, both older animals and especially young ones and sucking calves; after them come dogs. Horses, goats and cats suffer more rarely; pigs and sheep hardly at all. The complaint can, moreover, be transmitted to rabbits by inoculation. Ringworm is in general not a common or wide-spread skin affection, but seems rather to be located in certain byres and districts; and also in certain countries (England, Holland, France, Switzerland) is commoner than in others.

Many cases have been described in veterinary literature in which the tinea tonsurans of animals (cattle, dogs, horses) has been transmitted to man. Those affected have usually been the stable attendants who have been infected when cleaning or milking the animals, or rubbing them with ointment; or this has occurred in skinning them after slaughtering. Dogs seem to transmit the disease especially when being fondled. This infection in man may assume epidemic proportions; thus in 1840 most of the inhabitants of the Swiss village, Andelfingen, were infected by sick cattle. Köhler reports a case in which a newly stalled calf infected 21 cows, 5 calves, 9 horses, the herdsman and several other persons. Freytag describes a case where the three herdsmen belonging to a cow-byre all caught the disease, one of them conveying the infection to his wife and child. When transmitted in this manner to man, tinea tonsurans frequently causes an obstinate and tedious malady, very difficult to cure (Pusch). Cases have also occurred where the ring-worm of man has been conveyed to cattle, horses and cats.

Morphology and Physiology of the Trichophyton Tonsurans.—Microscopical investigation of trichophyton tonsurans enables us to recognize two elementary forms, viz., threads (hyphæ) and spores.

1. The *threads* or *hyphæ* are tender, elongated tubes, about four micro-millimètres thick, straight or undulated, dichotomous and possessing simple contours, sometimes articulated and sometimes not. They twist round the hair like nets, penetrate it and, when present in large numbers, form a perfect layer or bed of fungi (so-called mycelium).

2. The *spores* or *conidiæ* are small, spherical or elliptical cells, very refractile, and sharply defined, of about the same diameter as the hyphæ, from which after segmentation they develop. They are sometimes ranged side by side like a string of beads, and are usually much more numerous than the hyphæ, so that the latter are scarcely visible. These spores are very resistant and retain their power of germinating for more than a year.

The fungus vegetates especially in the hair-follicles and in the hair itself. The lower part of the hair-stalk is first enveloped in the fungoid mass, when a white, cylindrical mantle may be perceived by the naked eye at the base of the hair as a wing-shaped accretion. Later the fungus penetrates to the hair sheaths and roots, producing inflammation in the follicles which finally results in loosening and loss of the hair. Lastly it lodges in the interior of the hair itself, splitting it into fibres and rendering it brittle. The interior of the hair is often found so interpenetrated with conidiæ that nothing more of it is to be seen. The fungus seems to flourish best on dark skins and dark hair. The respective diameters of the spores and hyphæ are not the same in all animals, indeed on the same kind of animal they may vary considerably. Mégnin believed, therefore, that there must be two species, viz., a trichophyton tonsurans of horses and a tricophyton epilans of cattle; but differences in size are obviously of secondary importance and probably depend on variations in the source of nourishment. According to Grawitz, the tinea fungus liquefies gelatine after dissemination of seed more quickly than the favus fungus. When placed upon agar-agar, trichophyton produces a green turf-like covering, while the favus culture shows a stellate vegetation.

Symptoms.—In describing the morbid aspects of ring-worm we must first remark that a uniform description, fitting all cases, cannot be given, because the symptoms vary greatly according to the kind of animal and its breed, and even in the same animal, with the position and age of the eruption, and also according to its having been further affected by scratching, gnawing, rubbing, etc. In general we may cite as characteristic signs the following:—In the beginning clearly defined, round places, either hairless or studded with broken hairs, and mostly situated on the head, neck and extremities. These, which are at first the size of a lentil, extend outwardly and may attain the size of a crown-piece or more. The patches are mostly scattered, or run together and form larger ones. Sometimes the process ceases in the middle while spreading from the outer edge (so-called ring-worm, herpes circinnatus). Not unfrequently the eruption spreads over much of the body or over its entire surface, producing complete nakedness. On examining under the microscope hairs which have fallen or been pulled out, we find the hyphæ and conidiæ described above. The upper surface of the hairless patches shows either no inflammation at all, or else very varying degrees of it. With thick and more resistant hair, such as that of the horse, we find increased desquamation of the epidermis in the form of ash-grey, slaty or asbestos-like scales (tinea tonsurans maculosus). On finer skins we observe at first hyperæmia and swelling, in consequence of which the affected skin projects above the surface. Later on, vesicles are formed (tinea tonsurans vesiculosus), which quickly burst and are dried up to thick crusts and scabs, which in full-grown cattle are of different colours and leathery consistence, but in calves are a whitish grey colour like that of bread-dough, and also much fissured. The nature of these scabs is regulated by that of the hairy coat; being very thick where the coat also is thick, so that, as in cattle, they may be $\frac{3}{8}$ inch thick, while on parts less densely covered they are very thin. Beneath the scab is a suppurating surface, and after the crust has fallen off the places may heal of themselves.

The aspect of the disease in various kinds of animals is briefly as follows:—

1. **Tinea Tonsurans in Cattle.**—Small, round, sharply defined spots are found, covered with scabs or scales, standing either singly or in groups and usually upon the head

and neck, more rarely scattered over the whole body. These project more or less above the skin and are either bald or covered with greyish white scales, or with bristly hair. They may attain the size of the palm of the hand, and sometimes run into each other. On its outbreak, and also during the healing process, the eruption produces itching. The grey-white, asbestos-like scabs may become ⅜ in. thick upon black skins; on white ones they are thinner and more yellowish. Underneath we find a sticky, purulent fluid, as well as small and ulcerous hollows, which represent the distended, emptied and hairless follicles. Healing usually proceeds under the scabs, which gradually fall off, leaving small, slightly scurfy spots, upon which the hair slowly grows again. In other cases the skin remains very much thickened and arranged in folds (elephantiasis). The eruption lasts from six to twelve weeks. If, however, the normal course be disturbed by external influences, rubbing or scratching, fresh outbreaks occur within the healed patches, whereby, to some extent, a new inoculation takes place, and the ailment may be prolonged for six or twelve months or even longer.

In sucking calves we find the eruption chiefly about the mouth—so-called dough-mouth or mouth-mange. This affection, which is seen also in other sucking animals (lambs, kids, pigs) belongs probably in its essentials to tinea tonsurans, though it must be granted that a non-parasitic, impetiginous eczema may also occur which is confined to the region of the mouth. In most cases the evident contagiousness of the disease, as well as its clinical aspect, show it to be tinea tonsurans. Hahn has shown by his microscopical investigations into the nature of "doughy mange," that it is caused by tinea tonsurans, and published a description and drawing of the fungus. The phenomena of the eruption he describes as consisting in impetiginous round deposits upon the edges of the lips, ⅛ to 1 inch in size, lying on the visible surface and occasionally spread over the whole body, chiefly consisting of dry, bran-like, scaly matter, through which the hair projects, the latter being split up into fibres and destroyed by the fungus. In young animals the complaint is very obstinate.

2. **Tinea Tonsurans in Dogs.**—This disease chiefly affects the head and extremities of dogs, but sometimes spreads also over the whole body. It begins with round, sharply defined and solitary bald spots. Later we find isolated, clearly defined, hairless patches of round or oval shape and from ½ to 1 inch across. These are most numerous on the head around the lips and eyes and upon the extremities; not unfrequently they combine to form larger lobulated areas. According to the locality and age of the process we perceive varied morbid

signs upon these patches, which are either quite bare or covered with solitary hair-stumps. Very often the skin shows no symptoms of inflammation; and the tinea tonsurans occurs then with the aspect of alopecia or area celsi. But the spots are just as often incrusted with asbestos-like and dirty grey scales; in other cases with thicker scabs, by which the lower parts of the hair are stuck together. Beneath these scales the skin is from copper-red to brownish-red in colour and covered with numerous granules the size of a millet-grain (swollen hair-follicles). At times the affected portions project as much as ⅛ inch above the rest and present a real papulous surface, the skin round about being swollen and of well-marked tumid aspect. The older spots finally turn paler, are perfectly smooth and show but slight bran-like deposits. The symptoms in cats are similar (herpes maculosus).

3. **Tinea Tonsurans in Horses.**—Ring-worm is comparatively common among horses and may occur as an enzoötic affection among foals at pasture (R. Fröhner). It makes its appearance most frequently on the seat of the saddle, on the croup and flanks, but also upon the head. The several spots range in size from that of a sixpence or shilling to that of a crown-piece and form usually a fairly regular circle. The hair has fallen out or is broken off, and the desquamation of epidermis is considerable, reaching sometimes the stage of slight crusts. The surrounding hair can easily be pulled out, and that which grows again is usually darker in colour. Itching is generally absent, but may occur if the condition is very wide-spread (Schindelka). In isolated cases, herpes tonsurans vesiculosus may make its appearance, in the horse, upon the sheath and the inner surfaces of the thighs (Schindelka).

4. **Tinea Tonsurans in Sheep.**—We find the wool felted, and beneath it are bran-like, scabby parts upon the neck, chest, shoulders and along the back, and considerable itching is manifest. At the outset of the attack small tufts of wool stand out above the rest, and as these increase in number the fleece becomes very ragged in appearance. Schindelka saw ring worm develop in sheep which were kept in a place previously occupied by infected cattle.

5. **Tinea Tonsurans in Swine.**—This is of rare occurrence. It generally shows itself as herpes tonsurans vesiculosus

in single patches upon the back or the outer surface of the thighs. These patches are ring-shaped, slightly raised, and of about the size of a shilling; red in colour and covered with tiny blisters of the size of a pin's head, which do not cause irritation and which rapidly turn into little brown scabs. The bristles on the affected part do not usually fall out. Experimentally attempted infection of healthy animals was found unsuccessful (Schindelka).

6. **Tinea Tonsurans in Poultry.**—This shows itself in loss of feathers, and serious hyperæmia round about the papillæ.

Diagnosis.—Ringworm or tinea tonsurans is not very difficult to diagnose. The widely dispersed appearance of the eruption, its round shape, the loss of hair extending outwards from the circumference, the asbestos-like scabby deposits, especially on the heads of dogs (cheeks, lips, eyelids), the slight itch or entire absence of the same, the contagiousness of the disease and, finally, the results of microscopical examination, all help to make diagnosis certain. The evidence of the microscope may be obtained by extracting a few hairs from the edge of a bald place and examining their roots, or by inspection of the scabs, after previously softening them in a 10 per cent. lye of potash and isolating the hair-roots they contain. Viewed by the naked eye, the fungus is characterised by a white, mantle-like envelope on the hair-roots. Confusion with other skin complaints is particularly easy in dogs, in whom ringworm is often hard to distinguish from sarcoptic mange or from the squamous form of acarus eruption without the aid of a microscope. But these two latter complaints are accompanied by intense itching. In cattle, ringworm may sometimes be mistaken for a mercurial eruption, and *vice versâ.*

Prognosis.—This is so far favourable that cure always follows suitable treatment, if diligently and persistently carried out. In older cattle spontaneous recovery takes place as soon as the scabs have fallen off and the hair, which forms the chief nourishment of the trichophyton tonsurans, has gone with them. We have also noticed a few cases of spontaneous healing in dogs. Only in younger animals can the localisation of the eruption about the mouth cause difficulty in eating and finally death by inanition.

Therapeutics.—The prophylactic measures to be first adopted are, isolation of the affected animals, cleansing and disinfection of their stalls, destruction of all bedding of dogs, and instruction to the attendants respecting the infective nature of the disease. Then, in the next place, the scabs and scales must be thoroughly removed with green soap. Upon the patches thus cleansed the various anti-parasitic remedies may be applied, such as ointment of creolin, lysol, bacillol, carbolic, creosote, naphthol or tar (1 : 10); the different preparations of tar; creolin washes and creolin baths; grey, white, and red mercurial ointment (not on cattle), tincture of iodine (1 : 1–5 spirit), salicylic acid with spirits of wine (1 : 10), alcoholic solution of sublimate, or sublimate-salve (1 : 100), solution of arsenic, iodoform ointment; washing with blue-stone, alum, etc. In cases where the affection was not very wide-spread we obtained good and quick results with the very harmless creolin-salve and tincture of iodine, and also with another safe remedy, viz., alcoholic solution of salicylic acid. We applied them several times daily upon the affected places. Schindelka recommends an ointment containing salicylic acid and sulphur (acid. salicyl. ⅓ oz., sulfur. depur. 1 oz., sapo viridis and oleum Rusci āā 1⅔ oz., adeps suillus 7 ozs.), and a paste containing the same ingredients (acid. salicyl. 31 grs., sulphur precip. 2 drs., zinc. oxyd. and amylum āā ⅔ oz., vaseline, 1⅔ oz.). Baranski uses raw nitric acid (1 : 5 lard) rubbed into the skin three times. Behrens, Kramareff and others recommend a 10 per cent. creolin soap or creolin tincture of soap.

FAVUS. SCALL OR HONEY-COMB RINGWORM. TINEA FAVOSA.

Etiology.—The favus eruption described as scall or honey-comb mange, and also in poultry as comb-mange, fowl-mange and white-comb (tinea favosa), is caused by a mould-fungus discovered in 1839 by Schönlein and since named after him achorion Schönleinii. The disease occurs in dogs and cats, rarely in horses, but also in tame and wild rabbits and in mice. Among poultry the foreign breeds, such as Cochin-Chinas and Brahmas are specially attacked. The fungus lodges most easily upon the tender skin of young animals. It may be transmitted to man, and *vice versâ* (hence the name "inherited mange"). Cats appear to be mostly infected by eating mice suffering from favus. The favus fungus of man and the larger animals does not seem to be fully identical with that of fowls

and mice. The transmission of the poultry favus to horses, cattle or dogs, when attempted by Gerlach, was therefore unsuccessful. In the same way Schütz was only able to transmit its pure culture by inoculation to hens, but not to other animals (rats, guinea-pigs, doves, mice). Waelsch, basing his view upon his bacteriological experiments, is of opinion that the favus of men and mice is identical, but that its development is modified in each case by the nature of the nutritional material. Schindelka was able to convey the favus of fowls to other fowls but not to man or to cats. The favus of mice was transmissible to fowls, cats and dogs, but that of man not to mice, dogs or to old cats, but to young cats, fowls (discs as large as a sixpence on the comb), cattle (clinical appearances of herpes tonsurans maculosus; distinctly transmissible to man), horses and asses (similar appearances to those in cattle, and in a few cases, a sulphur-coloured scab formed after 2–3 months).

Morphological Notes on the Achorion Schönleinii.—The favus fungus forms round discs ("favi") or disc-shaped scabs on the skin, of different thicknesses and sunk in the middle, the so-called scutula ("small shields"), which consist mainly of fungoid elements and to some extent represent a pure culture of the fungus. Thus we find on inspection of these scutula a thickly felted layer of fungus (mycelium), formed of threads (hyphæ) and spores (conidiæ). These are morphologically exactly like trichophyton tonsurans. The threads are from two to five micro-millimetres in diameter, sometimes fine and long-drawn out, without partition, and at others, shorter, thicker, much ramified, segmented, occasionally swollen into club-shape with extremely granulated contents. Between these threads lie a great number of spores, at first cube-shaped, then round or oval and of equal diameter, which have developed from the threads by segmentation. Moreover the favus fungus penetrates into the hair-follicles and feather-papillæ, and finally into the interior of the hair and feathers themselves, which are thereby atrophied at the root and fall out. The threads and spores also luxuriate between the epidermal layers. Schütz obtained a pure culture of achorion Schönleinii of fowls upon peptone-gelatine broth at ordinary temperature and transmitted the same successfully to hens.

According to Quincke and Unna, at least three different

fungi can produce the aspect of the favus eruption (favus griseus, sulfureus tardus and sulfureus celerior). Others consider these to be merely "freaks" of a fungus which in its morphological and biological behaviour is very variable.

Symptoms.—1. *Favus in sucklings.* In these the favus eruption is characterised by dry scabs, which are outwardly greyish-brown to greyish-yellow or silver-grey, and internally range from white to sulphur-yellow, of a roundish disc-shape, concave in the centre, about the size of a shilling and up to $\frac{3}{16}$ inch in thickness. These scabs produce atrophy of the hair, with a flat indentation upon the skin. We find them chiefly on the head (ridge of the nose, cheeks, ears, forehead), on the belly, on the outer side of the thighs, and in cats round the claws and in the ear. St. Cyr describes them in dogs and cats as being crusts like mortar, cleft and fissured, of a grey or saffron-yellow colour and tarry consistence, which later scale off and have in the middle a saucer- or basin-like depression. At first they are perforated by a few hairs, which, however, fall off after a time. The skin beneath these crusts is thin and discoloured with blood. In dogs there is considerable itching, but only very little, or none at all, in cats. In horses the favus scabs may coalesce into long stripes the breadth of a finger. Sometimes animals suffering from favus emit a peculiar smell reminding one of cheese made from cows' milk. The course appears to be in general both benign and rapid. In one case Siedamgrotzky effected a cure after eight days' treatment, and St. Cyr in an average of eight to twelve days, the hair being renewed.

2. *Favus in fowls*, so-called white-comb, fowl-mange (tinea galli). The parasitic nature of comb-scab in fowls was discovered almost simultaneously by Leisering, Gerlach and Müller. The following are its phenomena. Small, white spots like mould appear upon the comb and lobes of the ears, which, gradually growing larger, unite to form a white coating over the whole comb. For several months this mouldy coating is restricted to the comb, and then changes into a thick scab, when the eruption attacks the skin of the head, quickly spreads itself over the neck, back, and finally over the whole body. The feathers become erect and fall out, and the bird is covered with scaly matter. It grows very thin, acquires a musty, mouldy smell and dies of exhaustion when the process reaches a certain stage.

Differential Diagnosis.—The differentiation of favus from tinea tonsurans is based mainly upon the presence of the disc-shaped scabs (scutula), which also, in contrast to those of tinea, are almost exclusively composed of fungus matter, and particularly of hyphæ, whereas such accumulations of fungus are not observed in tinea; in the latter there are also fewer hyphæ.

Therapeutics.—The treatment for favus is the same as that for tinea tonsurans. On removal of the scabs, apply the same fungus-destroying agents (creolin, sublimate, carbolic acid, iodine). Schindelka found that he got the best results with dogs and cats by the application of a 2–5 per cent. formalin paste; and with fowls, by frequent painting with 2–5 per cent. formalin solution. In fowls a spontaneous cure sometimes occurs. Healthy animals should be kept apart.

According to Ercolani, the favus fungus vegetates in the horny substance of the hoof, producing hollow places there (so-called seedy-toe) by disturbing the connection between the horny and sensitive layers, and being in this respect analogous to the onychomycosis favosa of man, which penetrates to the interior of the nails. For this reason Ercolani has called this hoof-complaint onychomycosis, and the fungus achorion keratophagus. Under the name of favus trichophytoides, Bodin describes a fungus which he found in two calves. It possessed the botanical characteristics of the favus fungus, but the pathological changes to which it gave rise were similar to those of trichophyton.

CONTAGIOUS ACNE, DERMATITIS CONTAGIOSA PUSTULOSA (CANADENSIS).

The so-called English or Canadian (American) Horse-pox.

Definition.—This complaint has really nothing in common with the true pox (variola) of horses, but represents a peculiar pustular and highly contagious exanthema with localised lesions, which, in slight cases, are accompanied by formation of simple blisters, pustules and scabs; in severe ones with more deeply penetrating suppuration and development of papules, ulcers and swelling of the lymph-vessels; but always without any disturbance of general condition, or without itching and benign, although its duration is often prolonged. According to Schindelka, it appears to be identical with the impetigo contagiosa of man (Kaposi), which is also an infective pustulous exanthema of the skin of the human head, of similarly benign

and typical course. Nocard considers contagious dermatitis to be a localisation of lymphangitis ulcerosa upon the skin and holds that the two complaints are identical.

Historical Notes.—English horse-pox, says Axe, to whom it owes its name of Dermatitis contagiosa pustulosa canadensis, was first introduced from Canada in 1877. From England it spread, through the export of English horses, over various parts of Europe, where it was observed in different places in 1879. Its identity with stomatitis pustulosa contagiosa, which was at first suspected, was later proved to be incorrect. But the exanthema appears to be of older date. In 1841–2, we learn from a description by Goux that an altogether similar, benign, very contagious pustular eruption, which affected chiefly the back, shoulders and croup, attacked the horses of a battery in the French army and affected them all within 14 days, with precisely the same symptoms as the so-called English horse-pox. Hering named the outbreak in question an "infectious tubercular eruption."

Etiology.—This epidemic skin affection is mostly transmitted from horse to horse by the rugs, bands, saddles, utensils and cleaning tools used. Consequently, the extent of the exanthema corresponds exactly to the superficial area of a rug, saddle or belly-band. For the same reason the positions occupied by these articles are the favourite seats of the eruption, which seldom passes on to the shoulders or front part of the breast. The head, neck and extremities, except in rare cases, have remained free. This evidently fixed contagion was declared by Axe to be of mycotic nature. Schindelka regularly found small micrococci in the pus and crusts, which possessed the quality of being easily stained by aniline; and Siedamgrotzky noticed the same in the form of diplococci. The skin affection could be transmitted by inoculation to horses, goats and rabbits; in the latter and in guinea-pigs Siedamgrotzky observed the occurrence in this manner of malignant œdema and septicæmia. Dieckerhoff and Grawitz made a pure culture, in the form of a very short bacillus, of the bacteria of English horse-pox, which they designate acne contagiosa. The fungus called "Acne-bacillus" by these two authors forms short rods (half the length of a tubercle-bacillus), in size about 0.2 microns, which divide by segmentation into elliptical or round balls, and, joining

together in twos and fours, form small chains. Both formative elements are very easily stained by watery fuchsin.

Bacteriological Notes.—When cultivated, the above-described bacillus forms very characteristic colonies and stale cultures, vegetating luxuriantly on blood serum. Its growth was most rapid at 98° Fahr., but half-an-hour's exposure to a temperature of 176°—194° killed it. When preserved dry, it retained its vitality for four weeks. Strange to say, it produced no putrefaction. It is quite sufficient to rub a few drops of water containing the bacilli upon the normally hairy skin of a horse to produce the typical exanthema. Inoculation succeeded with cattle, sheep, dogs and rabbits. Guinea-pigs were killed within 48 hours by merely rubbing the pure culture of the bacillus upon the sound skin, dying with the symptoms of septic poisoning. In grey mice a subcutaneous injection produced pyæmic phenomena. According to Nocard the bacillus is identical with that of lymphangitis ulcerosa.

Symptoms.—After a period of incubation lasting from 6 to 14 days (Schindelka), localised efflorescences appear on the seat of the saddle, on each side of the spine, which differ in character according to the intensity of the disease and the nature of the skin.

1. *In slighter cases* we first perceive round or oval prominences upon the skin, as large as a pea or hazel-nut, which either occur singly or in small groups, covering about the area of a man's palm and not very numerous. The hair above them is ruffled and bushy, the skin sensitive, swollen, and somewhat higher in temperature. These prominences are circumscribed inflammatory areas, and on closer investigation a varying number of isolated and rarely confluent vesicles and blisters will be found on their upper surface, which rise to the size of a millet-seed or even a bean, whose watery contents, at first clear, soon grow purulent (formation of pustules). In one or two days these blisters burst and change to thick, honey-coloured scabs as large as a sixpence or shilling, fastening the adjoining hairs together. About a week later both scabs and hair fall off, and circular spots, without either hair, pigment, scar or desquamation, remain, showing the normal epidermis, so that after about 14 days the entire exanthema has healed, having merely attacked the uppermost skin-layers down to the rete Malpighi or papillary body, without causing any disturbance of general condition. Only a slight swelling of the intermaxillary and of the upper cervical glands is regularly observed, which, however, soon passes away.

2. To this typical and slight course there are various and

frequent exceptions. The causes of these severer outbreaks are more intense infection, the confluence of several pustules and neglect of the eruption in not withdrawing the affected animal at once from work, but allowing the saddle, harness, etc., to increase the irritation and spread the disease. A consequence of such neglect is that the exanthematic process is extended, the inflammatory areas—at first solitary—increase to 50 or 60 in number and the pustules on such areas are multiplied four or five times. New prominences arise near the first ones, and around these again fresh inflammatory areas; the scabs grow very broad and may become larger than a crown-piece. The inflammation penetrates more deeply into the skin and even into the subcutaneous connective tissue, whereby hard, boil-like and larger nodules are produced, resembling gad-fly boils, which are very painful if touched and possess a suppurating centre, the contents of which may be squeezed out in the form of plugs of pus, leaving smaller or larger ulcerous cavities, which, by confluence, may become as big as a half-crown, and are later filled with granulations. From these larger purulent nodules inflamed lymphatics often radiate in various directions; indeed, suppuration of the neighbouring lymph glands, *e.g.*, the axillary glands, may even take place. This penetrative, purulent infiltration of the skin leaves scars behind it, differing in this from the superficial pustular lesion. Cure also is greatly delayed, and the process may last, with fresh outbreaks, for five to six weeks, or even two months. Even in these severest forms there is seldom a serious and general illness, and no real itching. In isolated cases the eruption finally settles in the extremities, where it may be complicated with inflammatory swelling of the joints (hocks).

Differential Diagnosis.—1. The *true pox* of horses (variola) has hitherto been observed only upon the pastern; and accompanied with feverish symptoms, which is not the case in Canadian pox.

2. *Farcy* may also show similar phenomena to those of the severer exanthema just described (ulcers, inflammation of the lymph-vessels, contagion). No blisters or pustules are formed, and there is no such recovery.

3. *Eczema impetiginosum and crustosum* and also *acne* closely resemble clinically the contagious pustulous exanthema. They, however, lack its great contagiousness, neither have such

large blisters and pustules been yet observed in eczema. Finally, the latter is marked by much severer itching.

4. Canadian pox may, at the beginning, be mistaken for *saddle* or *girth pressure*. This is the more lik ly since the introduction of the new pattern saddle into the German army, *sweat-eczema* now occurring far more frequently than formerly. The later course of the condition, which is very typical, removes all possibility of doubt.

Therapeutics.—Treatment is unnecessary in the milder cases, as they heal spontaneously. It is therefore only needful as a prophylactic precaution to isolate the sick animals and disinfect the stable utensils. The horses must also be withdrawn from work. In severer cases treat the open sores with antiseptics. Friedberger reports excellent results from the use of creolin (3 per cent.) and of carbolic acid ; Schindelka and Siedamgrotzky of 1 per cent. sublimate solution; Trasbôt of 1 to 2 per cent. solutions of blue-stone and sulphate of zinc. These also shortened the duration of the disease.

Farcy of Cattle (*Farcin du Bœuf*).—By this name a skin affection is known in France, which resembles the farcy of horses in the cord-shaped form of the swollen lymph-vessels, and is produced by a spore-making hyphomycetes (Nocard). This disease seems to have formerly been more common in France, and, according to Couzin, is stationary in the French colony of Guadeloupe. It has also been noted in Germany and Austria. It is a skin disease occurring on the extremities and belly and characterised by corded swellings along the course of the lymph-vessels (lymphangitis) and lymph-glands (lymphadenitis). By formation of metastases in the inner organs (pyæmia), or by general exhaustion, it usually leads to death after a course of years. It may be communicated to cattle and guinea-pigs by inoculation. In the latter, after intra-venous or intra-peritoneal injection, it produces symptoms closely resembling miliary tuberculosis.

Pseudo or False Glanders.—A number of specific fungoid infections occur among horses which present a clinical condition similar to that of glanders: (1) Nocard describes a glanders-like lymphangitis ulcerosa sui generis which occurs in French horses. It is caused by a specific bacillus and is characterised by ulceration of the legs. (2) A so-called yeast-lymphangitis, due to saccharomyces or cryptococcus farciminosus (yeast-fungus) which occurs among French, Italian, Egyptian, Japanese and Russian horses. It is characterised by the formation of ulcers and may gradually spread over the skin of the entire body. (3) A malignant mycosis of the skin and mucous membranes (buccal and nasal cavities, lips) in horses, with the formation of ulcers and subsequent necrosis, is caused by the mould-fungus hyphomycosis destruens equi (de Haan and Hooskamer).

III.—SKIN DISEASES CAUSED BY ANIMAL PARASITES. MANGE.

A.—*General Description.*

Definition.—Under the name of mange (itch, scab, scabies) we understand an infectious skin disease produced by the action of mange-mites; and without such mange-mites there can be no mange. In earlier times the name was used also for non-parasitic skin affections; thus it was customary to speak of "hunger-mange," "greasy-mange," etc., and before the mite was discovered the true mange was attributed to a dyscrasia of the blood. Among mange-mites in the narrower sense only three kinds are included: the sarcoptes, dermatodectes (psoroptes) and dermatophages (symbiotes), whereas the demodex folliculorum, which produces follicular mange, is usually not reckoned among the itch-mites. Each of these species of mite produces a different morbid condition in the various domestic animals, and each of the latter again possesses its special kinds of mite. The definition of mange must therefore be fixed in every individual case according to the respective species and kind of itch-mite, and we therefore speak more exactly of sarcoptic-mange, dermatodectic-mange and symbiotic-mange. Sheep and dogs are most liable to infection, and horses come next. Cattle, cats, pigs and poultry are more rarely affected. Mange, owing to its highly infectious character, is usually included among the epidemic diseases and should therefore have been treated in this category. But on purely practical grounds we have elected to rank it among the skin diseases, as in a text-book of special pathology, the description of clinical results is of the first importance. Certain forms of mange, such as foot and ear-mange, are of very trifling moment as infective complaints, possessing only a clinical interest.

Historical Notes.—The skin disease known in domestic animals as mange, scab, scall, etc., and in man as itch, is of great antiquity, and was obviously known to Moses as a disease of animals. The mange-mite was also long since discovered. According to Gerlach, it was first mentioned in the year 1174 by the Moorish physician, Avenzoar; and Fürstenberg states that the mites were described under the name of "Suren" in A.D. 1200, in the "Physica" of Saint

Hildegard. On our various domestic animals the mange-mite was discovered as follows :—

In 1672, on the cat, by Wedel.
In 1789, on the horse, by Kersting.
In 1809, on the sheep, by Walz.
In 1812, on cattle and dogs, by Gohier.
In 1846, on the pig, by Spinola.

The works of Gerlach and Fürstenberg have been in this department of most conspicuous importance, and among more recent authorities on the mange-mite we would indicate Robin, Mégnin and Zürn.

Morphology and Biology of the Mange-Mites.—The mange-mite belongs to the order of acarina (mites), division *arachnidae, family sarcoptes.* They are small, round to oval, articulated creatures, usually visible only with a hand-lens or microscope, 0.2–0.8 mm. in size. When fully developed they have four pairs of legs of five segments each, but only three pairs in their youthful stage, such limbs being furnished with suction or prehensile discs, bristles and claws. Their head, thorax and posterior part form one undivided body. From the head project prominent trophi, or feeding instruments, the jaws resembling saws, shears or bristles. As appendices to the outer skin we find scales, hair, spines, bristles, etc. The males are smaller than the females. We should exceed the design of a clinical text-book, were we to enter fully into the details of their structure and manner of life, and must, therefore, refer students to the special works on general pathology and the science of parasites. The females lay from 20 to 24 eggs at once, which hatch out in from four to seven days into larvæ, which, after three or four changes of skin, are capable of reproduction in from fourteen to seventeen days. Gerlach has calculated that in this manner about one and a half millions of sheep-mites may spring from a single female in 90 days. The females die in from three to five weeks after laying their eggs, the males live to be about six weeks old. The mites can exist isolated in moist air or on moist dung, etc., for a period of from six to eight weeks ; in empty stables, one to four weeks ; and in dry air two to three weeks. The eggs will keep for two to four weeks on a moist surface, but on a dry one only from four to six days. Both mites and eggs are killed in about an hour by a temperature of 104° to 140° Fahr. ; but a moderate

heat stimulates them, so that they become more active and propagate more rapidly in warm stables, under rugs and in summer time.

According to Gerlach, the mange-mites are most speedily killed by the following drugs:

By creosote in $\frac{1}{4}$ to $\frac{1}{2}$ minute; caustic potash (1 : 24), in 2 minutes; sulphurated potash (1 : 10) in $\frac{1}{4}$ to $\frac{1}{2}$ hour; oil of turpentine, in 5 to 9 minutes; tar, in 8 to 18 minutes; petroleum, in 7 minutes; tobacco (1 : 5), in 10 to 20 minutes; tobacco (1 : 10), in 2 to 5 hours; tobacco (1 : 50), in 4 to 10 hours; green soap, in $\frac{1}{2}$ to 1 hour.

According to the more recent tests of Müller, the several mange remedies stand, as regards their strength and rapidity of action upon the various kinds of mange-mites (and acarinæ), as indicated in the following table:—

Name of Drug.	Acarus.	Sarcoptes.	Dermatoryktes.
	Cease their movements entirely		
After being treated with :—			
Creosote 10%	at once.	at once.	at once.
Creosote 5%	in 6 minutes at most.	in 6 minutes at most.	almost immediately.
Carbolic acid 5%	in 5 minutes at most.	almost at once.	at once.
Sublimate 2%	at once.	in 15 minutes at most.	in 18 minutes at most.
Sublimate 1%	in 15 minutes.	in 40 minutes at most.	in 45 minutes at most.
Sublimate 0.66%	not after two hours.	not after 2 hours.	not after 2 hours.
Oxynaphthoë acid 5% (spirit)	——	in several minutes.	in several minutes.
Salicylic acid 10%	——	in a few minutes.	in about 15 minutes
Creolin	at once.	in a few minutes.	in a few minutes.
Ichthyol	in 25 minutes at most	in a few minutes.	in a few minutes.
Sulphurated potash 20%	in about 30 minutes.	in about 13 minutes.	in 22 minutes at most.
Sozojodol-mercury 2.5%	in 24 minutes.	in 95 minutes.	——
Arsenic 1%	not after 2 hours.	not after 2 hours.	not after 2 hours.
Decoction of tobacco 15%	in 85 minutes.	in 20-25 minutes.	in 35 minutes at most.
Wood-tar	at once.	almost at once.	almost at once.
Tar-liniment	at once.	in 5-6 minutes.	in a few minutes.
Peruvian balsam	in a few minutes.	at once.	at once.
Stinking animal oil	in 6 minutes.	almost at once.	almost at once.
Naphthol oil 10%	in 20 minutes.	in 2-3 minutes.	in 7-10 minutes.
Carbolic oil 10%	in 25 minutes.	almost at once.	almost at once.
Petroleum	in 95 minutes.	in 27 minutes.	in 42 minutes at most.
Naphthaline oil 10%	in 2 hours.	not after 2 hours.	not after 2 hours.
Oxynaphthoë-acid oil 10%	——	in 5-10 minutes.	in about 10 minutes.
Sozoiodol-mercury oil 20%	not after 2 hours.	not after 2 hours.	——
Mercurial salicylate oil 20%	not after 2 hours.	not after 2 hours.	——
Salicylic oil 5%	in a few minutes.	almost at once.	in 15-18 minutes.

From the foregoing it is plain that creosote, creolin, tar, animal-oil and Peruvian balsam act immediately and with almost equal strength upon acarus, sarcoptes and dermatoryktes. Carbolic acid, naphthol, petroleum, tobacco and sulphurated potash far more slowly, and are more fatal to sarcoptes and dermatoryktes than to demodex. On the other hand, sublimate and sozoidol-mercury poison the latter more quickly. According to Käppel, iodine in the form of Lugol's solution (1 : 80) kills dermatodectes in ten minutes, sarcoptes minor in six; and 1 : 40 kills demodex folliculorum in five minutes.

Experiments in killing the dermatocoptes of sheep by Brandl and Gmeiner give the following results: chloroform and carbon bi-sulphide, at once; 1 per cent. liquor cresoli saponatus, 3 minutes; 2½ per cent. creolin solution (Fröhner's mange-baths), 2½–4 minutes; 3 per cent. carbolic solution, 2 minutes; 3 per cent. carbolic oil and balsam of Peru, 8 minutes; 1 per cent. Lugol's solution, 10 minutes; tincture of iodine, 20 minutes; petroleum, 22 minutes; 1 per cent. sublimate solution, 50 minutes; 5 per cent. sulphuretted potassa, 34 minutes; 5 per cent. soda solution, 52 minutes; formaldehyde 1 : 5 water, 40 minutes; 2 per cent. salicylic oil, 40 minutes, etc. The germs remain alive after one hour's contact with the following substances: glycerine; soft soap; 20 per cent. decoction of tobacco; sulphuretted hydrogen solution; styrax tincture (1 : 5) chloroform oil; arsenic (1 per cent. solution); Helmerich's mange-ointment; grey and white mercury ointment; 10 per cent. sublimate ointment, etc. According to these figures, the remedies which are most efficacious in the treatment of dermatocoptes are: chloroform, carbon bi-sulphide, and liquor cresoli saponatus, the latter being best represented by lysol and creolin.

Species and Kinds of Mange-Mites.—The mange-mites as above described are divided into three species.

1. *Burrowing-mites, sarcoptes.*
2. *Sucking mites, dermatocoptes* (Fürstenberg); by Gerlach termed dermatodectes, and by Mégnin psoroptes.
3. *Scale-eating mites, dermatophagus* (Fürstenberg); by Gerlach called symbiotes, and by Mégnin chorioptes.

A. **The Sarcoptic-mites** bore themselves burrows under the upper skin and feed upon the cells of the rete Malpighi, which contain plasma. They also propagate in these burrows, producing an intense inflammation of the skin, and are characterised by their tortoise-like shape and extraordinary smallness (0.2—0.5 mm.), so that they can only be recognised under the microscope. Their heads are horse-shoe shaped and have two strongly-developed, conical, shearing jaws; the feet are short and stumpy, provided with pedunculated, tulip-shaped, prehensile discs, which in the male are attached to the first, second and fourth pair of feet, and in the female to the first and second only. As regards the subdivision of sarcoptes into varieties opinions differ. Fürstenberg has been guided therein more by morphological considerations, and Gerlach by biological. Attention has often been called to the great inconstancy of these varieties and to their different and remarkable deviations from

type (Zürn, Johne). We have adhered in the following to the sub-division made by Siedamgrotzky, in which there are two chief kinds : (*a*) the large burrowing-mite and (*b*) the small burrowing-mite.

(*a*) To the large burrowing-mites belong: 1st, Sarcoptes scabiæ of man, the horse, sheep, lion and tame rabbit ; and 2nd, sarcoptes squamiferus of dogs, pigs, sheep and goats.

(*b*) The small burrowing-mites, sarcoptes minor, occur in cats and in tame rabbits.

B. **The Dermatodectic Mites** live on the outer surface of the skin and, by means of their mouth-apparatus or trophi, suck blood serum and lymph from it, clinging the while to scales and hair. They also cause inflammation of the skin by pricking it. They are characterised by their size (0.5—0.8 mm.), so that on a dark background they may be recognised with the naked eye, and certainly with a magnifying glass, being identified by their long feet, long, pointed head, long, straight, piercing jaws, and lastly by their tulip or trumpet-shaped suction-discs, which are attached to articulated peduncles, occurring in males upon all four feet and in females upon the first, second and fourth pair of feet. According to Zürn, the dermatodectes are divided into :

(*a*) Dermatodectes communis, on horses, cattle and sheep.

(*b*) Dermatodectes cuniculi, the ear-sucking mites of the rabbit.

Even among the sub-varieties of dermatodectes there occur not unimportant diversities. Thus Zürn has described such an one on horses, and we ourselves have often noticed slight variations of structure.

C. **The Symbiotic Mites** inhabit especially the upper skin-surfaces of the extremities, feeding upon the younger epidermis scales, which they gnaw away. They are also moderately large (0.3—0.5 mm.) ; their outlines are, therefore, visible to the naked eye or with a magnifying lens. They are characterised by short, stumpy, conical heads, which are broader than long, an oval body slightly notched on its edges, long feet, tumbler-shaped suction-discs, which in males are found on all four feet, and on females on the first, second and fourth pairs. The male is also furnished on the hind part of the body with two prehensile organs, and the female with two cylindrical cones. Zürn thus sub-divides them :

(*a*) Symbiotes communis :

(1.) Symbiotes equi and bovis, which produce foot-mange and rump-mange.

(2.) Symbiotes ovis, which causes the foot-mange of sheep.

(*b*) Symbiotes felis, canis and cuniculi, producing ear-mange.

General Character of the Various Kinds of Mange and Classification of the Acarina as found on Various Domestic Animals.—The three varieties of mange-mites produce clinically different kinds of mange, which are due to the manner of life of the acarus.

1. *Sarcoptic-mange* begins by preference upon the head and those parts of the body which are least protected by hair, upon which, on sheep for instance, it exclusively occurs. It may spread from the head to the neck, and finally over the

whole body, as it does in horses, dogs and swine. On the dog it prefers the ears, the vicinity of the elbows, the lower breast, belly and inner surfaces of the extremities.

2. *Dermatodectic- or psoroptic-mange* favours the sheltered parts of the body, thus in horses the intermaxillary space, the inner side of the thighs, the neighbourhood of the sheath, the roots of the mane and tail; but may spread over the entire body when proper attention is not given to the skin. With sheep the dermatodectes mites seek first those parts which are protected by wool, and may therefore attack the whole body except the head.

3. *Symbiotic-mange* attacks chiefly certain parts of the body, thus in horses (and sheep) the lower portion of the feet, so-called foot-mange; in cattle around the root of the tail, so-called rump-mange; in dogs, rabbits and cats, the external ear-passages, so called ear-mange.

Upon the several domestic animals the following mites occur:—

A. On **Horses:** Sarcoptes scabiæ, dermatodectes communis, symbiotes equi; *principal-mange: Sarcoptic-mange.*

B. On **Cattle:** Dermatodectes communis, and symbiotes bovis; *principal-mange: Dermatodectic-mange.*

C. On **Sheep:** Sarcoptes squamiferus, dermatodectes communis, symbiotes ovis; *principal-mange: Dermatodectic-mange.*

D. On **Goats:** Sarcoptes squamiferus, dermatodectes and symbiotes; *principal-mange: Sarcoptic-mange.*

E. On **Dogs:** Sarcoptes squamiferus and symbiotes canis; *principal-mange: Sarcoptic-mange.*

F. On **Cats:** Sarcoptes minor and symbiotes felis; *principal-mange: Sarcoptic-mange.*

G. On **Pigs:** Sarcoptes squamiferus.

H. On **Rabbits:** Sarcoptes minor, dermatodectes and symbiotes cuniculi; *principal-mange: Sarcoptic-mange.*

Transmissibility in General of Mange to Animals and Man.— The following is what is at present known upon this point:

1. All the kinds of sarcoptes of animals may be transmitted to man and exist on him permanently. On the other hand, dermatodectes and symbiotes quickly die away on the human skin, or produce only slight signs of inflammation there.

2. From sheep, pigs, dogs, cats and man sarcoptes are transmissible to the *horse* and ass.

3. The sarcoptes of horses, sheep, goats and cats are transmissible to *cattle*.

4. The sarcoptes of goats may be transmitted to *sheep*.

5. The sarcoptes of man, the pig, cat, sheep and goat are transmissible to the *dog*.

6. The sarcoptes of the goat may infect *pigs*.

7. The sarcoptes of man may also infect *rabbits*. From the foregoing may be gathered that hitherto only the transmission of the various kinds of sarcoptes from one animal to another has been observed.

General Phenomena of Mange.—The disease develops only after previous infection. The period of incubation after transmission of the mites, that is, the time which elapses before any change is noticed upon the skin, varies according to the number of mites so transmitted. If only few, then four to six weeks may elapse before there is any manifest outbreak of disease; on the other hand this may occur within fourteen days. The infection is caused by direct transmission (straws, covers, utensils, brushes, etc., or by attendants). Poorly tended animals and such as are feeble and suffering from much scurf on the skin are peculiarly predisposed to infection by mange parasites.

The first symptoms provoked by the bites of the mites are a dotted redness (so-called stigma), nodules and blisters, an increased proliferation of epidermis and a deposit of crusts and scabs. From the very first these skin changes announce their presence by an intense itching, which, particularly in the night, under the rays of the sun or after sharp exertion, may become exceedingly acute. The animals then rub, bite and scratch the affected spots, and the mechanical irritation thus wrought very considerably alters the otherwise simple phenomena of the eruption. The hair falls out or breaks off, the wool grows shaggy, the skin discoloured with blood, and inflammatory processes are set up, which finally become chronic and cause permanent changes. Thus, at the climax of the disease we find the skin partly covered with impetiginous scabs and partly weeping and bloody, even ulcerously degenerated, thickened and creased.

Course.—This depends on various circumstances. First the kind of mite must be considered; whereas symbiotes produces mainly a localised mange, the eruption in sarcoptic-mange spreads readily over the whole body, though this occurs also with dermatodectes. Then the nature of the skin, the

degree of attention paid thereto, the quarters occupied by the animals, the season of the year, etc., are all of importance. In summer the greater heat causes a more obstinate course than in winter. Well-tended horses, such as military horses, will show only a slight degree of eruption for a long time. The ailment always decreases in sheep after shearing, because the mites are deprived of the sheltering wool. Finally, the intensity of the process may be greatly influenced by previous washing of the skin with suitable remedies. In general the course of mange is chronic. Spontaneous cures have not yet been observed; for the intermittent cases, in which the eruption considerably abates at certain seasons, cannot well be regarded as cures. When once the mange has spread over the whole body, general symptoms usually also appear. In consequence of the protracted irritation and loss of fluid, the animals go back in condition and growth, and may even perish of exhaustion and cachexia.

Diagnosis of Mange in General.—The recognition of mange as such can be based upon certain peculiarities of the eruption, apart from the presence of the mites. Among these are, first of all, its epidemic appearance and great infectiousness, the localised development of the eruption at the outset followed by very rapid spread. Pruritus is also much more marked than with other dermatoses. The locality affected by the mange likewise affords points for guidance. Thus, an eruption confined to the head, or spreading thence, with the above-named characteristics, indicates sarcoptic-mange; one restricted to the limbs or neighbourhood of the anus points to symbiotic mange; and, finally, one which seeks especially sheltered parts is likely to be dermatodectic-mange. But the surest diagnostic method is identification of the mite, which is best done by the microscope, when the scabs and crusts with their deeper layers must be taken off, cleared with a 10 per cent. solution of liquor potassæ, and then examined under a glass of low power. In the sarcoptic-mange of dogs the portion so taken off must go very deep, so that one must either scrape the skin with a sharp knife until it bleeds, or cut off a thin layer of the epidermis with a razor or scissors. Formerly it was recommended to bind a mange-scab on the human arm, when itching and slight inflammation would arise from sarcoptic-mange within twelve hours, and from dermatodectic within two hours. The microscopical test is, however, far more reliable.

The two larger mites—dermatodectes and symbiotes—may be rendered visible to the naked eye, or to a magnifying glass, by laying the scabs on black paper or a sheet of glass covered with a watch-glass and letting them be warmed in the sun-light, as under the influence of the warmth the mites move actively about or adhere to the watch-glass. To distinguish the various manges, see the earlier paragraph on the several forms of mange in different domestic animals.

Prognosis of Mange in general.—An estimate of the result of mange depends first of all upon the species of the mite and the extent and age of the disease. A sarcoptic-mange spread over the whole body, for example, is very difficult to cure in a horse, a fact which, in our opinion, has not been sufficiently appreciated. On the other hand, symbiotic mange in horses is easily curable. Wide-spread dermatodectic mange of long-standing is also very hard to remove from sheep, while in horses it may be easily combatted if of limited extent. Sarcoptic-mange in cats frequently ends in the death of the affected animal. Wide-spread sarcoptic and dermatodectic manges are therefore prognostically the most unfavourable. The same manges are more favourable when only local, especially symbiotic mange. The condition of the animal as regards nourishment is also an important factor in prognosis. Badly nourished or very young animals are hard to cure, being less able to endure the somewhat severe treatment. Weather and season also influence prognosis, in so far as with sheep, for instance, baths can be adopted only in warm weather without injury. The character of the hairy covering and the individual susceptibility to certain medicinal remedies must both be taken into account. There is much less chance of good results in treating thick-haired animals than with thinly-haired or shorn skins, and in sheep such treatment is usually fruitless until they are shorn. Many animals also are much more sensitive to certain drugs or forms of manipulation than others. Thus, carbolic acid cannot be used on cats without risk of poisoning. Cats, goats and poultry are moreover very sensitive to baths. Finally, the position upon the body of the mange is also an important consideration, as some parts are unsuited for the application of strong drugs, such as the neighbourhood of the eyes. The possibility also of fresh infection through unfavourable external conditions must not be overlooked.

General Remarks on the Treatment of Mange.— Mange can be cured only by the use of substances which destroy the mites. The mildest and also the weakest are the alkalies: potash-lye, green soap, the carbonates of soda and potash, also burnt lime, chlorinated lime, sulphate of potash, ammonia and the ammoniacal urine of cattle. As all these remedies also soften the scabs and epidermal-scales, they are mostly used with stronger ones, to help the latter to penetrate the skin. The various sulphur preparations also act very mildly, and in combination with the alkalies generate sulphuretted hydrogen, which has a fatal effect upon the mites. Still more intense is the action of tobacco, either as a lye or a decoction; also of arsenic and mercury, the latter in the form of sublimate or the less severe grey mercurial ointment. But most efficacious are the products of destructive distillation of vegetable and animal substances, above all creolin, lysol, carbolic acid, creosote, tar, benzol and stinking animal-oil. The form and combination of these in individual cases are dealt with later. Here we will raise only the following general points concerning the treatment of mange.

1. As a *prophylactic* precaution mangy animals must be *secluded* from sound ones as much as possible, and care be also taken that during treatment they are not afresh infected by other animals or by agents of transmission (covers, cleaning tools, straw, stalls). All these, as also the harness, and, in fact, the entire stable, should be carefully disinfected.

2. Before applying medicaments let the *skin* be *thoroughly cleansed* from all scales and scabs.

3. In using drugs for the destruction of the mites have regard to the *constitution and species* of the sick animal, and to any *pre-existing wounds* upon its skin. Feeble animals require gentler treatment, and there are drugs which may not be used on certain kinds of animal (mercury for cattle, or carbolic acid for cats). Sheep should not be rubbed or bathed with intensely poisonous mange-remedies immediately after shearing, because the skin then possesses greater power of absorption, owing to the numerous small wounds on it.

4. Those *places known to be affected* by the disease must be most *vigorously treated*; but treatment must extend, as a rule, to their non-infected surroundings, and in sheep should always cover the whole body, as every part is liable to infection.

5. The *application* of medicaments must be *repeated* after a certain interval, as most remedies can only destroy the mites,

but not their eggs, and from these latter fresh mites develop. In some circumstances, especially where the mange is very widespread and inveterate, a *third* application is advisable to kill any mites which may hatch out after the second. For the more deeply penetrative sarcoptic-mites even a third dosing with mange remedies does not suffice, but rather a repeated and even prolonged treatment is necessary in order to ensure a radical cure, especially with a sarcoptic-mange of long standing in horses.

Police regulations. According to the German police regulations, sarcoptic and dermatocoptic mange in horses, asses and mules, as well as dermatocoptic mange in sheep, are diseases which, since June, 1880, have been required to be notified.

Breach of warranty. According to the German civil law, mange in sheep constitutes a breach of warranty, with a period of warranty of 14 days.

B.—MANGE IN THE SEVERAL DOMESTIC ANIMALS.

a. *Mange in Horses.*

1. **Sarcoptic-Mange.**—This generally breaks out on the head, neck and shoulders and on the walls of the breast; but also, according to the mode of infection, may start from the withers, seat of saddle or surcingle, or on the hind legs and flanks. It begins with small, circumscribed bald patches, which gradually enlarge and may attain considerable dimensions. The eruption rarely covers the entire body, but rather keeps to the head, neck and shoulders. Its chief *symptoms* are an extraordinary itching, which is most intense at night, in hot sunshine or after heating of the body, and excites the animal to much rubbing, etc. If the attendant scratches the affected place it will evince lively signs of pleasure, which is shown by counter-pressure, curving of the back, whinnying and movement of the lips. The changes effected on the skin consist in small nodules, from which the hairs fall off, or are held together by a sticky fluid. Occasionally vesicles also are formed. As the disease proceeds, a very active desquamation of epidermis takes place on the nodules, with a gradual increase of scab-formation. Thus we have seen mange scabs nearly ⅜ inch thick, which consisted mainly of epidermal-cells, and in which the burrows of the mites could be traced in storeys. The skin also grows thicker and parchment like in sympathy with this inflam-

matory process, is crumpled into swollen folds, becomes cracked and fissured, discharging matter and pus, and has in places a sore and bruised appearance. In prolonged cases, and when the disease spreads over the whole body, the poor animals have a most wretched aspect, quickly grow thin and may even die cachectically.

The eruption is infectious to man, and attacks especially the hands and arms of the surgeon and attendants. In 1791, 200 hussars of one regiment were infected by their horses (Sik). On the other hand, in 1896, two horses in the 19th Prussian dragoon regiment caught mange from the men who groomed them. Other similar cases of the transmission by man of sarcoptic mange to animals have been observed and an etiological connection between the mange of man and the mange of animals has been assumed (Malkmus). Joest has noticed the transmission of sarcoptic mange from rabbits to asses and believes that, under certain conditions, it may be conveyed to horses.

According to official statistics, horse-mange is very rare in Germany as compared with sheep-mange. Thus, in the years 1887–1902, only about 9,000 cases in all were reported, a yearly average, that is of 600, and these occurred chiefly in the provinces on the Russian frontier. In Austria it is much commoner, no less than 12,553 cases having been reported between the years 1877 and 1887, of which 13.3 per cent. proved fatal.

The *diagnosis* of sarcoptic-mange is difficult to establish at first, because the mites are then not easily discovered. Rarely does one succeed in finding a single mite or its case, and at this stage the most careful search is often fruitless. The only important diagnostic symptom is therefore the extraordinary itching, which in the non-parasitic eczema (so-called nodular scabies or summer-mange) is by no means so intense. This sensation, combined with the slight skin-changes, led formerly to this being regarded as prurigo or pruritus, skin affections which, however, evidently corresponded in their chief symptoms to the initial stages of mange. In very long-standing cases of mange which have been much neglected, the mites are easily found.

The *prognosis* of sarcoptic-mange in slighter cases is favourable, but the reverse in severe ones. As regards its curability, sarcoptic-mange differs greatly from dermatodectic mange, being very much more difficult to cure and requiring in its older and severer forms a far more vigorous treatment, lasting

often for months. Relapse is, moreover, very frequent, and though this is usually regarded as the result of a fresh infection, yet it often occurs when all such possibility has been sedulously avoided.

Treatment consists first in cleansing the skin, softening the scabs with 5 to 10 per cent. carbolic or creolin-glycerine, and application of soft soap to facilitate their removal. The soap should be left on for about twenty-four hours, and, if necessary, the hair clipped off close. Having thus prepared the skin, apply the mange remedies in the form of ointment, liniment and washes. In slighter cases we commend Vienna tar-liniment, which is made up as follows: Rp. picis liquid.; sulfur. sublim. āā 1 $\frac{1}{10}$ lbs.; sapon. virid.; spiritus āā 2 $\frac{1}{5}$ lbs. Rub well into the skin with brushes, leave it on for from 6 to 8 days, renewing it wherever rubbed off, and then wash all clean, repeating the whole process twice or thrice. In most slight cases the result is permanent. When the mange is inveterate and widespread, it is well to alternate with other remedies, but often they all prove unavailing. Besides Vienna tar-liniment, we may recommend creolin liniment, prepared from creolin and green soap, āā 3½ ozs. spiritus, 1 lb. 8½ oz. In addition we may use carbolic acid or creosote (1 : 10 oil or spirits), arsenic-vinegar, sulphur ointment, styrax, solutions of creolin (3 per cent.), ditto of sublimate (1 to 3 per cent.), as also decoctions of tobacco (5 per cent.). But washing with sublimate alone does not seem very effectual, and we only obtained permanent cure after very many applications of a 2 to 3 per cent. solution, or after an addition thereto of a 15 per cent. tobacco decoction. Schindelka considers that the most efficacious remedies for mange in the horse are: sulphur ointment (15 per cent.) and creolin ointment (4 per cent.).

2. **Dermatodectic Mange.**—This begins with sharply-defined spots, somewhat larger even from the first, and mainly situated on the sheltered parts of the body, such as the roots of the mane and tail, in the maxillary space, on the sheath, on the udder and inner sides of the thighs. We have also seen it in other places, such as the withers, back and croup. The earliest phenomena are nodules, which represent an inflammatory reaction of the skin against the injuries caused by the bites of the mites, and from which a lymph-like fluid oozes, and dries into a scab. Increased desquamation of epidermis also takes place, with falling-out of the hair. The deposits of scales and

scabs may in course of time become as thick as the finger. Later, the entire skin gradually thickens, takes on a leathery appearance, and crumples into folds. The itching sensation is here also somewhat considerable, and serious inflammatory conditions of the skin may result from rubbing, and lead to penetrative suppuration and bloody sores.

The *differentiation* of dermatodectic mange from the sarcoptic form is based—in addition to identification of the mite—upon its preference for sheltered parts of the body, the formation of clearly-defined areas, and its slower spread. We must admit that, in cases spread over wider areas, its clinical aspect very much resembles that of sarcoptic-mange. Any confusion with eczema squamosum (scaly scabies) and mane-mange may be avoided by observing the previous acute itching of dermatodectic mange and the sharply-defined outline of its areas—this apart from microscopical investigation.

Recovery is in cases of dermatodectic mange usually much more rapid than in the sarcoptic variety, and with the relatively milder remedies used we sooner obtain the desired result. *Treatment* with creolin or carbolic soap, or with creolin or carbolic glycerine, in the proportion of 1 : 10, generally suffices; or Vienna tar-liniment may be applied. The latter is, however, less suitable when the scabs are very thick. On an average even severer cases may be cured in from fourteen days to three weeks. Only with stallions, which show a formation of deep folds on the upper margin of the neck, does a cure appear to be difficult, or even impossible (Trasbôt).

3. **Symbiotic-Mange.**—This variety, which has also been called foot-mange, is characterised by a great preference for the feet. It usually occurs in the pastern, whence it spreads to the fetlock and to the surfaces of the hind and fore shins, and may even rise to the hock, knees and elbows. In rarer cases it ascends to the shoulders, neck and lower and upper thighs. Its development is remarkably slow, so that a distinct extension is perceptible only after a lapse of months. Its *symptoms* are first seen in a repeated and, we might say, spasmodic stamping (this is often the earliest sign) and even kicking, especially at night, also in rubbing and gnawing of the fetlocks. If we examine these, we find abundant desquamation of epidermis and loss of hair, followed later by the appearance of scabs, cracks and fissures; the skin becomes brittle and rough, thickens and, in prolonged course, reveals horny, papillary

proliferations. The *diagnosis* of this foot-mange is guided by its position, the desquamation of epidermis and the violent itching. This latter item distinguishes it also from ordinary eczematous greasy heels. Only in exceptional cases, when it spreads over the whole body, can it be mistaken for sarcoptic-mange. *Treatment* consists in the application of creolin or carbolic-glycerine, carbolic soap or tar-ointment (1 : 10). These remedies suffice to effect a cure in a short time.

Mégnin describes symbiotic mange as "intermittent mange," and maintains that it appears in winter and vanishes in spring, but that during this period the mites remain alive, but live during the warmer season upon the more abundant hair secretions, whereas in winter they are compelled to attack the skin itself. According to Schwarz, the frequent wounds upon the pasterns are caused by the horses' own restlessness when suffering from foot-mange, as a result of which their pasterns often become entangled in the halter-rope.

b. *Scab in Sheep.*

Statistical Notes.—All three kinds of mite infest sheep, but dermatodectic mange is by far the most important form of the disease. How serious its ravages sometimes are may be shown by the following figures. In 1884 an official report showed that in Germany nearly a quarter of a million sheep suffered from scab, and in Alsace-Lorraine the proportion affected reached nearly 61 per cent. of their entire sheep. Statistics show that, from 1885–1888, in Prussia, there were 183,000; in Bavaria, nearly 100,000; and in Wurtemburg over 60,000 cases of mange among sheep. The loss in Prussia alone was nearly 2,000 animals. In the years 1887–1902, the entire number of sheep suffering from mange was close upon two millions. Nevertheless, mange among sheep is on the decrease in Germany; the figure fell from 145,000 in 1891, to 97,000 in 1892, and to 65,000 in 1893. The districts in which the disease is at present most common are: Cassel, Brunswick, Hanover, Osnabrück, Hildesheim, Stade, Minden, Upper Hesse, and Luneburg. An idea of the loss in wool may be gained from Fürstenberg's statement that, in the Greifswald district alone, it runs into an annual sum of 100,000 marks (£4,875); this means that the annual loss to the whole of Germany amounts to many millions of marks.

Pathogenesis.—The mode of transmission of the mange-mite is usually direct; an infection by some carrying agent being

less frequent. Transmission occurs most easily when shorn sheep suffering from scab are turned out among a flock still unclipped. It takes place also more freely in warm temperatures, in summer, in the stable, or when the animals lie together, as the mites are then much livelier. With regard to the biological condition, Brandl and Gmeiner have ascertained that, in places in which infected sheep have been put, the mange-mite retains its power of conveying infection for as long as three weeks in warm weather, and one week in cold weather.

Phenomena of Dermatodectic Mange.—The favourite spots for the dermatodectes mites are those covered with wool, while the more naked parts, and especially the head, are neglected. They usually attack the flanks and root of the tail, spreading thence over the back, sides, neck, shoulders, etc. The eruption always develops from small spots which, according to the mode of infection, are either solitary or spread in varying numbers over the body. The progress of the development is as follows: On dividing the woolly coat we see at first flat nodules the size of a millet-seed, of pale yellow or reddish colour, which have been caused by the bite of the mite, and, according to the number of these, are either found singly or united into flat prominences as large sometimes as a half-crown. The skin round about is reddened, and the whole region at first painful when touched. Blisters soon appear on the top of these nodules and small pustules, which burst and dry up. At the same time active desquamation of epidermis sets in, which, in combination with the more abundantly secreted fatty sweat, forms a yellowish deposit, and when conjoined to the exuded matter of the blisters and pustules, produces often a very thick, hard, yellowish-brown mange-scab, beneath which the parasites flourish. Upon shorn skins this scab assumes an aspect resembling parchment. The wool is loosened at the root and glued together by the fluids discharged from the nodules, and as the wool grows, the mange-scab is also lifted up with it. The entire fleece gradually becomes shaggy and flaky, larger or smaller tufts rise above the general surface, which finally fall or are rubbed off. The separate wool-fibres are lustreless, pale in colour and easily pulled out. The skin below the scabs has lost its lustre and smoothness, is rough, dry, crinkled, thickened and covered with fissures and chinks, and, in consequence of much rubbing, is ulcerated.

With these local changes of the skin there is also very

intense itching; indeed, this is usually the first symptom of sheep-scab. The animals rub, scratch, gnaw, pinch and pluck at the affected spots, shake and roll themselves about, sink their backs, and on being scratched evince the liveliest pleasure, quivering their lips, scraping involuntarily with the hind feet, shaking their heads, clashing their teeth, wagging the tail violently and pressing up against the hand which rubs them. This itching is most severe in warmth and towards evening. The constant rubbing hastens the fall of the wool and causes more or less serious inflammatory and ulcerous affections on the skin. If the mange spread far, the animals lose flesh, owing to the persistent irritation, loss of fluids and hindrance in feeding and rumination. Feeble animals consequently often perish (hydræmia, general cachexia). The course of the disease always shows considerable aggravation in autumn and winter, being very appreciably checked by shearing and washing. Finally, the tenderer skins of the finer breeds seem to suffer more intensely than those of ordinary sheep.

Diagnosis.—For the identification of scab the following points are of importance. 1. *Proof of infection*, or of the extended presence of the disease in a flock. 2. *The characteristic itching* and restlessness on the part of several animals when inspecting the flock. We must, however, point out here that if an animal be vigorously rubbed by anyone examining it, no itching may be observed, but only signs of pain, a fact often noticed at the commencement of an outbreak of scab eruption. Also that the shepherds know how to suppress all expressions of relief and pleasure by firmly holding the sheep during its examination by the surgeon, and can also temporarily remove the itching sensation by the use of certain remedies. 3. *The condition of the wool*; a flaky, shaggy fleece, easy extraction of the wool and the presence of bald patches are suspicious signs. 4. *Careful examination of the skin*, especially about the loins, root of tail, back and shoulders. These parts should be thoroughly palpated with the finger-tips, and the skin exposed by parting the wool. Any discoloration of the wool by remedies previously applied by the shepherds, so-called grease spots, must be carefully noted. 5. Diagnosis can only be rendered positively sure by *microscopical proof of the presence of the mites.*

Differential Diagnosis.—The following may be confounded with dermatodectic mange:—

1. So-called *rain-rot*, an eczema produced upon the skin by persistent rain and which, like scab, may spread through a whole flock. The rapid healing of this eczema on return of dry weather or removal to cover, the absence of itching, combined with the method of its origin, all facilitate differentiation.

2. *Other parasitic skin eruptions*, especially those caused by lice. In such cases a careful examination of wool and skin may prevent confusion.

3. *Excessive accumulations of wool-fat*, by which, under some circumstances, the hairs are glued together. Apart from the absence of itching, the skin will be found in such cases to be perfectly clean, smooth and lustrous.

4. Wilful *erosions of the skin*, by caustic remedies, may be recognised partly by the colour of the substance used, and partly by the intense uniform slough, though sometimes it may not be easy to distinguish them from mange-scabs. But the later course and microscopical investigation will furnish sufficient data for a sound opinion.

Therapeutics.—In the treatment of sheep-scab a distinction is drawn between healing by inunction and by dipping. The first is only a provisional operation, intended as a preparation for the latter, or performed, for instance, in winter, because the other cannot then be undertaken. It consists in rubbing of local scab areas with mite-destroying ointments or liniments (carbolic soap, carbolic oil, creolin-liniment, grey mercurial ointment, tobacco-juice, petroleum). This rubbing treatment does not suffice for a radical cure, for the simple reason that only the visibly-affected spots are anointed, while smaller centres, spread perhaps over the whole body, remain untouched. Objection has been raised from many sides to the use of grey mercurial ointment on the ground of its poisonous action, which may even extend to the lambs of the ewes thus treated. It is wiser, therefore, to replace it by other and safer remedies. For dipping purposes numerous sheep-dips have been introduced, on the value of which divers opinions are held. The following are the chief of these :—

1. The so-called **Walz's dip,** the oldest known bath for mange, was introduced by the discoverer of the dermatodectes mite, and consists of 4 parts of quick-lime, 5 parts of potash (or 60 of wood-ashes), 6 parts of stinking animal-oil, 3 parts of tar, 200 parts of ox urine and 800 parts of water. This is

the cheapest of all sheep-dips, but is by many considered too weak, as severe cases or those of long standing cannot be healed by it; moreover, it dyes the wool brown. It is therefore only appropriately applicable as a preparation for one of the other stronger mixtures.

2. **Gerlach's Scab-dip** consists first of a preparatory bath of 2 parts of potash, 1 part of quick-lime and 50 parts of water; and the mange-bath proper of a 3 per cent. tobacco decoction. According to Roloff, this latter is better in a 5 per cent. strength. This dip of Gerlach's has generally been recognised as very effective, especially since Roloff's modification. It is, however, troublesome to prepare, and the preliminary bath affects the hands injuriously.

3. **Zündel's Scab-dip** consists of 3¼ lbs. of commercial carbolic acid, 2¼ lbs. of quick-lime, 6½ lbs. each of soda and soft soap mixed in 57 gallons of water, this quantity sufficing for 100 sheep. This very cheap dip (costing about 2s.) may be improved, according to Kayser, Ostertag and others, by using a 2 per cent. decoction of tobacco instead of water (about 11 lbs. of tobacco to 57 gallons of water), whereby, it is true, the cost is trebled, but the effect greatly augmented. This dip is then a combination of those of Gerlach and Zündel. A similar mixture was previously recommended by the Prussian and Bavarian Governments. According to this 16½ lbs. of home-grown tobacco were boiled for half an hour in 55 gallons of water, and 2¼ lbs. each of pure carbolic acid and potash added to the warm liquid (cost price, 11s.). This very effective dip is, however, troublesome to prepare, and may lead to poisoning of the sheep dipped therein. Instead of a decoction of tobacco-leaves, the tobacco lye might be used. Kayser recommends the substitution of nicotina, a species of tobacco-lye.

4. The **arsenic-dips** introduced by **Tessier and Matthieu** have met with an extraordinary diversity of appreciation; some taking exception to their poisonous character, others praising their great efficacy. But in many cases it seems that their poisonous action can be traced to careless application, especially just after shearing. The numerous small abrasions caused by clipping must all be thoroughly healed before dipping the animals, so that this should never be done until 8 to 14 days after they are shorn. Tessier's mixture consists of 3¼ lbs.

of arsenic and 22 lbs. of sulphate of iron, with 22 gallons of water. As modified by Trasbôt, Tessier's dip is made up of 2¼ lbs. of arsenic, 11 lbs. of sulphate of zinc, 17½ ozs. of aloes (all the weights quoted in this section are avoirdupois) and 22 gallons of water; this formula is largely used in France. Matthieu uses alum instead of sulphate of iron (2¼ lbs. of arsenic, 22 lbs. of alum and 22 gallons of water). This arsenic and alum dip has been widely introduced in Würtemberg and Bavaria. Kehm employs 17½ ozs. of arsenic, 13¼ lbs. of alum and 22 gallons of water. It is important to observe that the arsenic is previously completely dissolved in boiling water to prevent any corrosive action. Putscher has also (verbally) recommended these arsenic-dips, but always precedes them with a cleansing bath of soft soap, and in severer cases has almost always found three dippings to be necessary, with an interval of nine days between each.

5. The **creolin-dip** recommended by **Fröhner** is carried out as follows:—If possible, the sheep are first shorn. This is not absolutely necessary with the creolin-dip; indeed, scabby sheep may be treated with good results in their wool and without any injury to the staple. After shearing, let all visibly scabby spots, especially the loins, back and neck, be rubbed for several successive days with a creolin-liniment, until the scabs are softened. This creolin-liniment consists of one part of creolin, one of spirit and eight parts of green soap. This embrocation forms an integral portion of the treatment (preparatory inunction). The sheep thus prepared are dipped twice in a watery solution (2½ per cent.) of creolin, an interval of seven days being left between each bath. The solution contains 1½ gallons of creolin to 55 gallons of water for 100 sheep, and is prepared by simply pouring the creolin into warm water 86° Fahr., and stirring. Each bath lasts three minutes, and after each such dip the animal must be vigorously scrubbed all over with brushes and then plunged again for a few seconds into the bath. The method of holding the sheep and all other manipulations are the same as in other forms of scab-dipping. There should be no undue haste in the process, and careful oversight given to the workmen employed. The cost of each such bath is 8s., as against 11s. for the carbolic acid and tobacco-bath. A third dipping will be necessary only if the instructions given have been imperfectly obeyed. This creolin-dip is distinguished from other

scab-dips by its certain and reliable action, and more especially by its non-poisonous nature and its cheapness (5½ gallons for 1s. 2½d.). Moreover, the wool is neither dyed by the creolin, nor in any way damaged, as is always the case with tobacco and arsenic. Of 750,000 sheep dipped in creolin in Prussia during the years 1888–1901, 85 per cent. were cured.

The **method of washing** in scab-dipping is as follows:—After shearing, allow 3 or 4 days to elapse, which interval must with arsenic dips be extended to 8 or 14. The temperature of the bath must be about 86° Fahr.; for each sheep reckon from ½ to ¾ of a gallon of liquid. All the sheep in a flock must be dipped, including the apparently healthy, which should be taken first. The body must be completely immersed with exception of the eyes, nose and mouth, and remain so for three minutes. Have the dipping-tub placed near the fold, in a cool, shady place, or, better still, partly sunk in the ground, and close at hand must be the scrubbing-tub. Each sheep must be dipped by two men, one holding all four feet, and the other the head, carefully protecting the eyes by pressing a thumb over each, and then plunging the animal in back downwards. Immediately after the bath the animal must be lifted into the scrubbing-tub by two other men, vigorously brushed, rubbed and its fleece run through with the fingers, especial care being devoted to the back, loins and rump. Then plunge once more into the bath for a moment or two, and turn it loose into a *clean* stable or sunny pen-fold. Avoid the risk of its getting wet by rain, as this not only brings danger of chill, but washes out the fluid and lessens its healing effect. The operation must be undertaken only in warm weather. After the bath let all the stabling and shelters previously used be disinfected. A week later repeat the scab-bath, and again, if necessary, in fourteen days. Two dippings are in all cases indispensable. During the interval any freshly discovered scab-areas must be locally treated; and even when the whole dipping process is over, the flock should be carefully observed for some time. Finally, avoid as far as possible the routes and pastures previously used. In view of recent events, it is as well to point out that creolin and other medicaments will not do away with scab in sheep. The treatment must be combined with a strict and conscientious observance of the police regulations.

In place of creolin, other similar remedies may be employed. Such are, liquor cresoli saponatus and bacillol. Brandl and

Gmenier recommend a single 1 per cent. bath with liquor cresoli saponatus, but this must certainly be too weak a solution to be efficacious in general practice. Maisel recommends a 2 per cent. lysol solution and Paszotta and others a 2 per cent. bacillol solution. Akaprin, recommended by Regenbogen, has not proved a reliable remedy in sheep-scab.

Sarcoptic-Mange in Sheep.—According to Gerlach's and our own observations, this occurs exclusively upon the portions of the head which are not covered with wool (called, therefore, *head-mange of sheep*), and rarely attacks the legs. Its favourite spots are the lips, corners of the mouth, ridge of the nose, chin, round about the eyes, also the cheeks and outer side of the auricles. Upon these we find firmly adherent, grey scabs, about $\frac{3}{8}$ to $\frac{5}{8}$ inch thick; and there is usually great itching. In warm weather the eruption spreads almost all over the head, retreating on the advent of a colder season. The ailment causes no further harm, and may be cured by creolin-liniment, or washing with creolin or strong tobacco-decoction. Artificial transmission to the woolly skin of a merino-sheep only produced a fleeting inflammation of the skin. A sarcoptic mange much resembling the mange of goats was observed by Roloff in sheep with fat buttocks (scabs as hard as stone and like scale-armour on the head and other parts of the body). Creosote or carbolic acid in glycerine were found to be the most efficient among mange remedies.

THE FOLLOWING ARE THE DIPS APPROVED OF BY THE BOARD OF AGRICULTURE AND FISHERIES IN ENGLAND.

Sheep Scab Order of 1905, No. 6,879. Prescriptions for sheep dips approved by the Board for sheep-scab.

Quantities for 100 gallons of bath.

1. Lime and sulphur:—Mix 25 lbs. of sulphur with $12\frac{1}{2}$ lbs. quick-lime. Triturate with water until a good thick cream without lumps is obtained. Transfer this to a boiler capable of boiling 20 gallons by the addition of water; boil and stir during half an hour.

The liquid should now be of a dark red colour; if yellowish continue the boiling until the dark red colour is obtained, keeping the volume at 20 gallons. After the liquid has cooled, decant it from any small quantity of insoluble residue, and make up the volume to 100 gallons with water.

2. Carbolic acid and soft soap:—Dissolve 5 lbs. of good soft soap, with gentle warming in 3 quarts of liquid carbolic acid (containing not less than 97 per cent. of real tar acid). Mix the liquid with enough water to make 100 gallons.

3. Tobacco and Sulphur:—Steep 35 lbs. of finely-ground tobacco (offal tobacco) in 21 gallons of water for four days. Strain off the liquid, and remove the last portions of the extract by pressing the residual tobacco. Mix the whole extract, and to it add 10 lbs. of flowers of sulphur. Stir the mixture well to secure an even admixture, and make the total bulk to 100 gallons with water.

NOTE.—The period of immersion in these dips should not be less than half a minute.

NOTE.—At present (1908) all sheep-dips used in Great Britain must be "approved by the Board of Agriculture." A large number of proprietary dips have been so "approved."

Symbiotic-Mange in Sheep.—This mange, which is also called the *foot-mange of sheep,* attacks the lower parts of the feet, and may even ascend to the testicles or udder. It is marked by itching, desquamation of epidermis and formation of scab.

c. *Mange in Dogs.*

Occurrence.—This is a very common disease among dogs, especially in large cities. In Berlin nearly 10 per cent. of all sick dogs suffer from mange. Among 70,000 sick dogs treated at the Berlin Veterinary College in the years 1886–94, 6,225 were suffering from sarcoptic mange (Fröhner). In Münich, in 1889, 16 per cent. of the medical cases treated were cases of mange (Friedberger). In Cologne, more than 50 per cent. of the dogs suffer from mange (Sticker). The percentage of mange at Stuttgart (Hoffmann) and Dresden (Müller) is the same as at Berlin. Dog-refuges, and breeding and training establishments have undoubtedly much to do with its dissemination.

Symptoms.—The mange of dogs (sarcoptes squamiferus) usually appears on the head, viz., on the ridge of the nose, at the root of the ears and on the eyebrows; also the belly, lower chest, the region of the elbows, the root of the tail and the paws. But the eruption may begin on other parts of the body, *e.g.*, on the skin of the genitals. The mange generally spreads rapidly, so that after a month the whole body may be affected. Its aspect, however, varies much according to the power of resistance possessed by the skin.

Its first phenomena resemble flea-bites, being red spots, which are visible chiefly on the unpigmented skin, as well as on the tenderer skin of the belly and inner sides of the thighs. The surface is also often diffusely reddened owing to much rubbing and scratching. Upon the site of these, papules, varying in size from a millet-seed to a lentil, are soon perceptible, which may change into vesicles. These latter are often numerously scattered all over the body and, on bursting, leave large weeping places behind (so-called weeping mange). In other cases the process is quite dry, and turns to abundant desquamation of epidermis (so-called dry mange). More rarely the vesicles change into pustules, and this particularly on the

finer skin of the lower belly and inner thighs, which pustules often show a dark centre the size of a pin-prick, is the seat of a mange-mite. Vesicles and pustules soon dry up into greyish yellow crusts, while the upper skin scales off very much and the hair falls out, so that large bald patches appear. The skin itself thickens later, becomes wrinkled and creased and cracks are opened in it by the animal's rubbing. Most frequently we find the skin of head and neck folded into numerous wrinkles, which give the animals a very characteristic appearance. These skin-changes are accompanied by very intense itching, which increases in a warm room, near a fire or after any bodily exertion, and keeps the dog in a state of constant unrest. If we scratch or rub the affected part the animal shows much delight and scrapes involuntarily with one or other of its feet. If the eruption lasts long and spreads much, the dog grows very thin, and feeble animals may even perish.

Diagnosis.—The most important diagnostic signs of sarcoptic-mange are: Excessive itching, the frequent commencement of the evil on the head, its rapid spread, the ease with which it can be communicated to other dogs and even to men, the wrinkled appearance and baldness of the skin, the accumulation of scurf and scabs and the marked preference of the eruption for the ears, lower chest, lower belly, elbows and inner thighs. The surest of all diagnostic guides, discovery of the mite, is, however, generally very difficult. Owing to the depth at which it hides, it is effectually protected and can only be found by removing the deeper layers of skin with the scabs. To this end we must either scrape off the scabs with a sharp knife until they bleed, or remove smaller superficial portions of skin with scissors. Or we may try to tempt the mites from their lairs by heat, placing the dog in the front of a fire or covering it with a rug. In other cases, especially where the crusts become very thick, the mites are more easily demonstrable. As a last diagnostic resort we may bind a mange-scab on the human arm. Upon dead animals the mites may easily be discovered.

Differential Diagnosis.—Sarcoptic-mange is most frequently confused with the eruption of demodex. Both are infectious, both are accompanied by formation of nodules and baldness of skin and also with much desquamation of epidermis. The chief differences are that in sarcoptic-mange the itching

is far more intense and that it is easier to cure; also that the demodex eruption usually displays a typical pustular character, and finally, that the parasites are easily found. An infectious skin-eruption, accompanied by intense itching and the other symptoms described, may always be regarded as mange when the results of microscopical investigation are negative. Sarcoptic-mange may also be mistaken for certain itching, papular eczemas. But the mistake is soon removed by the rapid healing of the latter. Widespread eczemas, which permanently vanish on a single application of mange remedies, must be presumed not to be sarcoptic-mange. In the latter disease we also notice its communication to man, which fact may sometimes be of importance in distinguishing it from a simple eczema. Hundreds of men in Berlin, for instance, are every year so infected.

Therapeutics.—The *prognosis* in dog-mange is much more hopeful than in the mange of horses. It must not be forgotten that the cure of long-standing and widespread mange in dogs is not always so easy, and that feeble and debilitated dogs frequently are unable to bear the treatment, and perish under it. Certain preliminary precautions are more necessary in dogs than in other domestic animals, as they must be prevented from licking off the drugs applied by tying up their mouths, muzzling them or covering them with leather wraps. The anti-parasitic remedies should also not be applied immediately after shearing, which, in long-haired animals, is indispensable. They should also not be laid on to sore places of large area.

The following drugs are to be recommended in the mange of dogs:—1. *Cresol:* We generally use a cresol-liniment which, according to the intensity and area of the eruption, we make of equal parts of cresol and green soap, with ½ to 10 parts of spirit. This liniment we rub in daily (in extensive mange one-third of the body daily); and a cure results on an average in two or three weeks. Also cresylic alcohol and spirit (1 : 10–20) and cresol-soap, as well as 2½ per cent. creolin-baths may be used. Creolin, being non-poisonous, when properly diluted, possesses one great advantage over other mange-remedies.

2. *Tar:* Usually in the form of a tar-liniment (tar and soap equal parts, spirit, q. s.), and so applied that only one-third of the body is anointed at once. After remaining three days on the body, the liniment must all be washed off, and the dressing

repeated. Cases of moderate severity are thus cured after three to four dressings.

3. *Creosote,* which is best mixed with olive or rape oil in the proportion of 1 : 10–15, and applied as above. With this remedy care must be taken to guard against poisoning.

4. *Carbolic acid,* as 2 to 5 per cent. carbolic oil or carbolic glycerine. With all three remedies it is well to leave a longer interval between each dressing, to prevent the risk of carbolic poisoning. By way of precaution a sulphate might be administered (sulphate of soda, to generate the harmless phenylsulphonic acid in the blood). Among other remedies we can recommend *Peruvian balsam,* which is particularly good for very delicate dogs and for treating the surroundings of the eyes. On the other hand, we have had only poor results from using naphthalin, which, moreover, smells badly. According to Siedamgrotzky, Tolu-balsam is also unreliable. Lysol (liquor cresoli saponatus) and bacillol are similar in their action to creolin. Brandl and Gmeiner recommend a cresol liniment (Aqu. cresol. 50, soft soap and alcohol $\overline{aa}$ 25). Finally, we may mention as more or less useful remedies, benzol, naphthol, oxynaphthalin, ichthyol, endermol (nicotinum salicylicum) and septoform (Rabe, Friedberger). Benzine and petroleum are almost ineffectual. Epikarin, recommended by Frick, does not possess any advantages over the other remedies for mange; on the contrary, it is unreliable in its action, and, in bad cases, quite useless (Regensbogen and Müller). Brandl and Gmenier consider it useful in treating young and delicate dogs. Rievel considers peruol (peruskabin), as recommended by Regensbogen, of little use. Diet must be regulated during the whole time of treatment, and chills carefully avoided.

Mange is so widely distributed and the danger of communication to man is so great that the necessity for police supervision grows every day more necessary. It should either be included in the category of notifiable diseases, or it should be dealt with by strict local police regulations.

Symbiotes on the Bodies of Dogs.—The symbiotes-mite of dogs, first discovered by Hering in 1836 and by him named "sarcoptes cynotis," is found sometimes in the purulent secretions of the otitis externa and also in the external ear-passages. That it can be brought into etiological connection with otitis is more than doubtful. Zürn and others believe in such a connection, basing their opinion on the processes in the ear-mange of cats. Nocard says that he has observed epileptiform spasms and deafness.

Sarcoptic-mange in the Fox.—McFadyean has frequently observed sarcoptic mange in foxes in England, the disease having been probably transmitted by dogs. The animals were very thin and almost hairless. It was found possible to transmit the disease to dogs.

d.—*Mange in Cattle.*

1. **Dermatodectic-mange.**—The favoured spots for this most frequent form of mange in cattle are the sides and hollow of the neck and the root of the tail. Thence it spreads over the back, the lateral thoracic walls, the neighbourhood of the horns, shoulders and even over the whole body. Its chief *indications* are: Nodules, exudation of fluid, increased desquamation of epidermis, loss of hair, great itching and a formation of very dry, greyish-brown scabs. The skin at last becomes thick and leathery, rough and wrinkled. Sometimes the disease is more acute in winter, with a corresponding improvement in summer. As the eruption spreads over the body the animals grow thinner and sometimes die. Röll observed this form of mange in buffaloes. It appears that dermatodectic-mange occurs also along with symbiotic-mange. *Treatment* is the same as for other animals; thus, for example, creosote and decoctions of tobacco have been found very effectual.

2. **Symbiotic-mange.**—This form, which is also known as "*rump-mange,*" occurs mostly in the hollows about the anus, as well as around the root of the tail, but may spread all over the body (back, neck, inner thighs) if due attention be not given to the skin. Such cases, nevertheless, are rare. It may also spread downwards to the lower parts of the feet (*foot-mange*). Its *symptoms* consist in deposition of scales and scabs, loss of hair and itching, which may last for years without further incommoding the animal or any serious spread; it is only very slightly infectious. *Treatment* is rarely needful; as with horses, the milder mange-remedies suffice (carbolic-soap, creolin-ointment, tar-ointment).

Sarcoptic-mange.—This is extremely rare in cattle and is manifestly communicated by mangy horses. It represents therefore no independent form of mange and demands no further description, the symptoms being essentially the same as in horses. Neumann even doubts its existence.

NOTE.—There is no longer any doubt about sarcoptic-mange affecting the ox, numerous cases having been recorded during recent years.

e.—*Mange in Goats.*

The **Sarcoptic-mange** of goats, which is the usual form of the disease in these animals, attacks at first only the lightly-haired places, more particularly the head (lips, ears, nose), but quickly spreads over the whole body. It is characterised by deposits of grey, bran-like scales, and of bluish-grey scabs, like those of fish, which are hard and fissured. The skin becomes thickened, cracked and wrinkled, grey and parchment-like, and shows bald patches, which gradually enlarge, so that the animals are often quite naked. There is also intense itching. Goat-mange does not seem to be at all rare, for in ten years there were nearly 12,000 cases in Austria alone. It is also frequently fatal to goats, and Wallraff noted 500 deaths out of 1,000 animals affected. The mite is readily transmitted to man, and in one village Wallraff found nearly every family infected. Roloff also transmitted the mange to short-haired sheep (Somali) and to those with smooth wool and deficient in sweat (fat-rumped sheep). As regards *treatment*, baths should be avoided, as goats cannot bear them well. Use therefore creolin-ointment, tar-ointment, sulphur-salve, carbolic glycerine, etc.

By French observers (Mégnin and others), both a dermatodectic-mange (external ear-passages) and a symbiotic-mange (on the trunk) have been identified in goats.

f.—*Mange in Pigs.*

The **Sarcoptic-mange** of pigs (Sarcoptes squamiferus) is seen first on the head, especially in the hollows of the eyes, on the eyelids and cheeks, around the ears, and spreads thence to the neck, back, inner sides of the thighs and finally over the whole body. At the outset we find these places covered with bran-like scales, blisters and pustules, and later with strong, whitish-grey or silvery scabs up to $\frac{3}{8}$ inch in thickness, so that the animals look as if sprinkled with guano. Their skin is thick, wrinkled, very hard and firm. Their bristles also are loosened or fallen out, and sometimes stuck together in tufts. Beneath the scabs are mites, which, however, are difficult to find upon the living animal. *Treatment* consists in softening the scabs with soft-soap or glycerine, and in rubbing them with a tar or sulphur ointment.

In young pigs mange is sometimes called "sucking-pig rust," and generally represents a non-parasitic eczema (compare p. 487). That this "rust," as Sohnle believes, is always caused by sarcoptes-mites is improbable. Attempts to transmit the mange of swine to dogs, sheep and goats have failed (Brandl and Gmeiner, Albrecht).

g.—*Mange in Cats.*

Symptoms.—The position most favoured by mange (sarcoptes minor) in cats is the head, which usually is alone attacked and especially the ears. At first red dots appear, also papules and blisters on the skin, which is then covered with small impetiginous scales. The hair becomes rumpled and partly falls out, and the upper skin desquamates freely. This scurfy matter, combined with the secretions from the papules and blisters, grows gradually to solid, greyish-yellow mange-scabs which measure up to $\frac{3}{8}$ inch in thickness and contain a mass of mites. The underlying skin becomes hard and stiff, thickens and lies in folds. The eyelids often swell, and catarrhal and suppurative inflammation of connective tissue sets in. Itching is considerable, and the animals keep scratching themselves with their paws, shaking their heads and rubbing them against hard objects. Their general condition also is much deranged, even when the mange is strictly confined to the head. They are dull, grow thinner and not unfrequently perish. This eruption may also spread to the neck and entire body ; when the paws are first affected, then the loins and flanks. In such cases death is much more likely to result. Transmission of cat-mange to mankind may also occur.

Therapeutics.—There is no great need for vigorous treatment ; but baths, washings and embrocation of the whole body with oily remedies should be avoided. Carbolic acid, as being specially poisonous to cats, must never be used. As the best and most harmless remedy we recommend Helmerich's sulphur-ointment. This is made up as follows : sulphur 2 parts ; carbonate of soda 1 part ; lard 16 parts. We have always effected a cure with this salve, provided the animal were not too much run down in condition. Not quite so harmless as this sulphur ointment is the otherwise excellent Peruvian balsam, after a limited application of which on the head we have at least a few times noted grave signs of brain irritation, and even decided maniacal symptoms with subsequent stupor and even death. The latter we have seen also after inunction

with rancid cod-liver oil, a remedy recommended by some. Styrax appears to be somewhat less dangerous.

A *Sarcoptic-mange* on **beasts of prey of feline species** (lions and leopards) has been observed by Delafond and Bourguignon, and in more recent times by Johne. But this was not provoked by the sarcoptes minor, but by the larger burrowing-mite, sarcoptes scabiei communis. Its signs were at first circumscribed mange-patches on the breast, belly, back, etc., which speedily spread over the whole body, which became scabby, encrusted and almost hairless. The animals quickly grew thin, lost appetite and finally died. Sprinkling with Zündel's mange-dip resulted in fatal carbolic acid poisoning. The treatment adopted by Johne was an application of Peruvian balsam (1 : 3 spirit), the animals being introduced into a short and narrow cage, through the bars of which they were sprayed with Peruvian balsam and then vigorously scrubbed with brushes attached to long iron staves. After five such treatments, with intervals of from eight to ten days, they were perfectly cured.

The so-called *ear-mange of cats* is caused by **symbiotes auricularis.** The animal keeps shaking its head, holds it on one side and betrays symptoms of otorrhœa. We have found Peruvian balsam with an equal part of glycerine an excellent remedy, the ear-passages being previously cleansed.

h.—*Mange in Rabbits*

1. **Sarcoptic-mange.**—The mange eruption produced by sarcoptes minor closely resembles cat-mange. It attacks chiefly the head (particularly nose, lips, brow), and is accompanied by formation of crusts and scurf, itching, thickening of skin, loss of hair, etc. Along with the head the ends of the extremities are generally also affected; but the mange may spread all over, when, as a rule, the rabbit quickly grows thin and dies. A mange caused by sarcoptes scabiei also occurs on rabbits and sometimes covers the whole body and is communicable to ferrets and guinea-pigs. As with cats, the *treatment* consists in rubbing with sulphur-ointment or Peruvian balsam.

2. So-called **ear-mange,** induced by symbiotes and dermatodectes cuniculi, is a somewhat common condition, consisting in inflammation of the covering of the external ear-passages, which develops into very thick layers of stratified, yellow-brownish, dry and fissured scabs, which sometimes completely fill up the ear-passages so that the auricle appears quite rigid. Zürn also observed violent inflammation of the

middle ear and a dissemination of the dermatodectes mite over the whole surface of the skin. *Treatment* consists in softening and removing the scabs, and application by means of a brush of creolin-glycerine or carbolic-glycerine, or else creolin-oil or carbol-oil (2–5 per cent.), to the covering of the exterior ear-passages. Both of these forms of mange, with the same localisation, occur in ferrets.

FOLLICULAR MANGE.

Occurrence.—This eruption is most common on dogs and cats, rarer on pigs, cattle and goats; but the acarus-mite has been seen on sheep, the Sambur stag and finally on bats, rats, field-mice and earless marmots. It is also very frequently found in the comedones or dilated sebaceous glands of man, but without causing any injury. In isolated and rare cases follicular mange has been transmitted from dogs to men.

Natural History.—The sac-mite, hair-sac mite, or *acarus folliculorum*, or *demodex folliculorum*, forms a species of the family of dermatophili, in the order of acarina, class *arachnoidea*, and belongs therefore to the group of arthropodes (articulated animals). It was first discovered by Henle and Simon in 1843, in the comedones of man, and, in the same year, by Tulk on dogs. Since then the following varieties have become known :— acarus folliculorum canis, cati, suis (demodex phylloides), bovis and hominis.

In its fully developed state the body of the demodex folliculorum of dogs is worm-shaped and lancet-like, but in pigs and cattle it more resembles the form of a laurel-leaf. It is about $\frac{1}{75}$ to $\frac{1}{100}$ inch long and divided into three segments, head, thorax and abdomen, the two latter, however, being joined into one piece. The head is lyre or horseshoe-shaped and consists of various chitin-parts, which may be subdivided as follows :— 1. The front head, which forms the foundation of the entire apparatus for mastication ; 2. a pair of upper jaws or mandibles ; 3. a pair of clasp-like lower jaws or maxillæ ; 4. the movable, many-jointed feelers or pedipalpi ; 5. a single stiletto-like formation, being the so-called oral-valve. There are also two eyes in the form of unpigmented nodules on the sides of the anterior head. The thorax is short and bears in its two free edges four pairs of stumpy movable limbs, each made

up of three parts, hip, thigh and terminal member, the latter of which has five chitin-pieces which give it rather the appearance of a paw. The lower surface of the breast displays a central ridge with four transverse ridges on each side. The long abdomen is also transversely striated, serrated along its edges and pointed behind. The young acarus has only three pairs of feet, is smaller and narrower and has no rings about its abdomen, and is hatched from spindle-shaped or oval ova. They pass through three stages of skin-formation before reaching maturity.

According to the investigations of Brandl and Gmenier, the transmission and distribution of follicular mange occurs, not only directly, but also indirectly, by the conveyance of the virulent contents of the pustules by intermediaries. When there is much aqueous vapour in the atmosphere the acari may live as long as 3 days; in dry air they die within 24 hours.

Pathological and Anatomical Notes.—Microscopical examination discovers the mites chiefly in the sebaceous glands, and especially round about the evacuatory exits, also in the upper parts of the hair-follicles, there being an average of 30 to 60 in each sebaceous gland. This number may rise to 100 or even 200, so that the glands or their orifices bulge like voluminous bags. Along with these we often find a mass of eggs and larvæ. The mites lie in the glands almost without exception with their heads downwards. Around these bulging glands inflammation sets in, and white blood-corpuscles ooze out, so that the peri-glandular tissue becomes purulently broken down and, with the gland, is changed into a purulent pustule. But these acne-pustules develop only when the mites are present in large numbers. When less numerous we perceive atrophy and falling out of the hair.

The skin of the pig shows the most serious changes. Here several pustules run together and form large abscesses, which may contain as many as 1,000 mites, and cause deeply penetrating ulcers in the skin as large as half-a-crown. If the eruption lasts long, the skin becomes sclerotic and often from three to four times its normal thickness. Mange mites are found not only in the skin of dogs suffering from this disease, but are also present in the aural meatuses, and in the preputial and conjunctival secretions (Horneck).

Symptoms in Dogs.—The eruption caused by demodex folliculorum on dogs is very widespread and occurs in all breeds. Of 70,000 dogs treated in the Berlin dog-hospital in nine years, not less than 1,200 (nearly 2 per cent.) had been attacked by acarus. Many animals show individual differences as regards the intensity and course of the complaint. With some the eruption remains for months confined to the same spot, while with others it spreads quickly over the whole body. Singularly enough, the complaint can only with difficulty be transmitted from dog to dog by inoculation, as compared with its evident contagiousness. Of many such attempts at transmission which have been made, almost all, except a few observed by Hauber, Guinard and others, have resulted in failure. Neither can the disease be thus communicated to other animals, but a few cases of spontaneous transmission to man have, on the contrary, been observed. Its further spread upon the same animal results from rubbing and scratching, whereby the pustules are emptied and their contents, as it were, sown over the body. Connected with this fact is also its quick dissemination after the earliest inunctions with medicaments. The aspect of the malady varies according to the numbers of mites present in the sebaceous glands.

1. *Squamous form of the acarus-eruption.*—In many cases, besides the loss of hair, we only find an increased desquamation of epidermis, the surface of the skin being perfectly dry and showing no signs, or only very slight ones, of inflammation (eczema squamosum). This form occurs especially around the eyelids. At the same time the hair has mostly fallen out, leaving a bare circle about the eyes, the skin is slightly reddened and covered with cast-off epidermal scales. This limited appearance with such slight reaction of the skin makes the diagnosis of follicular-mange somewhat difficult, because its chief characteristics, the acne-nodules and acne-pustules, are absent. For this reason every case of the hair falling out, accompanied by slight skin-inflammation on the eyelids, should be microscopically examined for acari. This kind of eruption is sometimes spread in patches all over the body, but usually without any itch. Its aspect has, however, an extraordinary resemblance to tinea tonsurans. Occasionally a darker pigmentation of the bald places is a characteristic sign of the disease.

2. *Pustular form of the acarus-eruption.*—More frequently the eruption manifests itself as an intense inflammation of the

skin, with characteristic formation of papules and pustules, corresponding to the position of the sebaceous glands. This form may also occur in circumscribed patches, especially on the head (ridge of nose, lips, eyelids) and the paws, but may also spread all over the body. But it favours the head, throat, neck and inner sides of the limbs. At first the affected skin is swollen, higher in temperature and hyperæmic. The hair is ruffled, and scattered nodules reveal themselves, from a millet to a hemp-seed in size, which gradually change into bluish-red or purely suppurative pustules. The skin thickens, often to double its normal density, and is partially covered with greenish-yellow, sticky and purulent exudation, mingled here and there with blood ; and partially with dried-up, greyish-yellow or blood-coloured crusts. The hair has fallen in places and can be very easily pulled out, when purulent matter often adheres to its lower ends. If the skin be laid in a fold and slightly pressed, blood is discharged and, on pressing more heavily, often a number of plugs of pus will follow. The degree of itching varies much in different individuals, being sometimes severe and sometimes almost absent, though in general it is much slighter than in sarcoptic-mange. If during its further progress the eruption be not altered by the animal's scratching and rubbing, the skin will gradually dry up, but will remain thicker and disposed to lie in folds, showing several hairless or very scantily haired patches, on which, in the latter case, the hair is ruffled and strikingly lustreless. The skin is also covered with thick layers of blue-grey epidermal scales and scabs, between which are fissures and cracks which easily bleed. It thus resembles the bark of a tree or an elephant's hide, and the head particularly reminds us of a wart-hog (phacochœrus). The surface of the skin is also rough and covered with lumpy, hard, nodular excrescences, from which further pus-plugs may be squeezed. The unpigmented parts of the skin are of copper-red colour, or else display upon a slightly reddened ground bluish-red, knotty protuberances (hence the earlier name of "red-mange"). We find in the upper skin-layers, corresponding to the situation of the suppurated sebaceous glands and hair-follicles, pitted hollows, with proliferations of connective tissue resembling papillæ. In spite of these serious changes, the animal's general condition is often very little disturbed, there being nothing beyond a mild chill and slight loss of flesh ; its appetite, indeed, is usually increased, so that it often seems to be ravenous.

This aspect of follicular-mange is, however, gravely altered if the animals gnaw or rub much. The affected spots are then changed into hairless sores, which secrete pus, or even into genuine skin-ulcers. At the same time enormous swellings of certain parts, such as the head, round the eyes and neck, arise as a result of inflammatory œdema of the subcutaneous connective tissue. In this condition the animals rapidly grow thin, emit an unpleasant, sweetish-putrid smell and present a wretched appearance, when they die either from exhaustion or of pronounced septicæmia.

Symptoms in Pigs.—The phenomena of the skin-complaint produced in pigs by demodex phylloides are, according to Csokor, protuberances, varying from a grain of sand to a hazelnut in size, out of which small tumours at first develop, which grow larger and become at last broad skin-ulcers. As an intermediate stage, pock-like lesions are mentioned by some observers, which may give rise to confusion of this eruption with swine-pox. Only the softer-skin parts are attacked, such as the vicinity of the snout, neck, lower breast, flanks, groin, belly, and especially the inner sides of the thighs, while the upper part of the head, back and outer sides of the limbs escape. The matter which can be squeezed out of the tumours contains from 50 to 100 mites, with their various larval conditions and cast-off husks, while the larger abscesses shelter from 500 to 1,000. This mite is twice as broad as the demodex folliculorum of dogs. The eruption is much more contagious than that of dogs. According to Rieck, d modex mange is more common among swine than is generally supposed. Single pustules are frequently found after slaughter, especially in the flexor surfaces of the knees.

Symptoms in Other Animals.—Grimm and Oehl have noted cases in **cattle** in which the skin of the entire body, excepting only the head and extremities, was sprinkled with nodules from the size of a pea to a hazelnut, which were most abundant on the neck and shoulders. These nodes were hairless, smooth of surface, and from them a thick, tough pus could be pressed, in which a very large number of demodecidæ could be found. Other parts showed only redness, slight swelling, loss of hair and desquamation of epidermis. Oehl states that cattle recovered spontaneously, but a cure was hastened by rubbing in a 10 per cent. creolin ointment.

Upon **goats** Bach noticed about 40 such nodes, from a pea to a cherry in size, with dough-like, greyish-yellow contents, and especially

on the shoulders and about the flanks. These nodules contained each up to 1,000 acari. There was moderately severe itching and great loss of condition. A treatment extending over 30 days proved to be of no avail.

In **cats** the demodex eruption is seated chiefly upon the head (nose, eyes, ears); the clinical aspect is similar to that in dogs.

Rabbits.—Pfeiffer observed an epizoötic occurrence of acarus-mange in rabbits in China, which took the form of an eczema squamosum, and made its appearance upon the eyelids and ears. It not only destroyed the eye and the outer ear, but gave rise to violent inflammation of the inner and middle ears, and, in some cases, to inflammatory conditions of the brain which resulted in death. The parasite was considerably smaller than the mange-mite found in dogs.

Diagnosis.—In many cases the evidence of the characteristic bluish-red pustules, combined with localisation of the malady upon the head or extremities, suffices for diagnosis even on a superficial examination. It certainly can only be secured in the squamous form of follicular-mange by microscopical investigation, which must be carried out by thinly spreading some matter squeezed from a pustule upon the object-glass of a medium-power microscope. If there are no pustules, scrape the skin superficially and place the scrapings, or the roots of a few plucked-out hairs, under your glass. The ailment may be *mistaken for:* 1. non-parasitic, impetiginous eczema and traumatic suppurative dermatitis; 2. for the pustular exanthema of distemper in dogs; 3. for sarcoptic-mange; 4. for inflammation of the eyelids (Blepharitis); 5. for tinea tonsurans; and 6. for non-parasitic acne and furunculosis.

Prognosis.—In a few cases it is said that a spontaneous cure may occur, but these are rare. In general the forecast must be unfavourable. When widely diffused, the malady has so far been seldom cured, even after months of persistent treatment. On the other hand, apparent cures, followed later by relapse, have not unfrequently been published as genuine. In any case it is necessary carefully to watch dogs that seem to recover, for weeks and months, before we can be sure of the result, and it is very easy to be deceived in this matter. Even when the eruption is locally restricted, we must be very cautious in making a forecast. A cure is then easier, but the complaint may notwithstanding spread very rapidly as a direct result of the treatment. We would always warn owners from the very first of the great obstinacy of the disease. When the acarus

eruption is widely spread, it is better to kill the dog at once. But the individuality of the various animals much affects prognosis.

Schindelka does not altogether confirm the view that acarus exanthema is incurable. He has succeeded in curing it in over 50 cases, but considers that recovery is dependent upon the devotion of much time and patience on the part both of the owner and of the veterinary surgeon. In the squamous form, a quicker, easier, more certain recovery is possible than in the pustulous form. Before commencing the applications, all deposits upon the skin should be removed by means of baths; sulphur is afterwards used as an anti-scorbutic.

Therapeutics.—The number of remedies in use for follicular-mange is very great, and well illustrates the helplessness of therapeutists. So far the following have been recommended: The various carbol-preparations, creolin, benzol and petroleum, Peruvian balsam and styrax, mercurial mixtures, cantharides-ointment, sulphuret of potash, naphthalin, naphthol, strong coal-tar oil, nitric acid, tincture of iodine, Lugol's solution, sozoiodol, ethereal oil of juniper berries, sulphur-ointments, etc. According to our own experience, those which have best stood the test of experiment are: Peruvian balsam, cantharides-ointment in combination with sulphuret of potassium, sublimate-ointment and also inunction with concentrated creolin and carbon bisulphide.

1. *Peruvian balsam* is especially recommended by Siedamgrotzky, who cured a dog which was affected all over after a two-months' daily anointing with it and carefully squeezing out the pustules, curing not only the local sores, but the entire eruption. We also have cured slighter cases, but only after prolonged treatment, in one instance extending over 58 days. In another case of a badly diseased animal we obtained a great improvement, which looked like complete cure, after 16 weeks of treatment, but on cessation of this the eruption soon reappeared. Very long and persistent treatment with Peruvian balsam is required to effect a complete cure. The remedy, moreover, is very costly, and renders the symptoms at first more severe.

2. The plan adopted by Brusasco in Turin consists in washing the affected skin with a solution of *sulphuret of potassium* (1¼ drams in 1¾ pints of water) and subsequent inunction with dilute cantharides-ointment. We also achieved complete

cure in very advanced cases in a relatively short time. Care must be taken not to touch the conjunctiva of the eyes with the cantharides, or suppurative panophthalmia may set in, which may even prove fatal. We would therefore advise treatment of that region with Peruvian balsam. The process is best carried out by first clipping the animal (if the eruption be widespread) and bathing it completely in sulphuret of potassium (say 7 ozs. to 15½ gals. of water for a large dog), and on the *next* day rubbing one-third of its body thoroughly with the dilute cantharides-ointment, and another third part on the next and following days. One or two days later wash it all well off with the solution of sulphuret of potash; and then, after a few days' pause, recommence anew, continuing thus until a complete cure is effected. That even this plan may fail is proved by the case of a Pomeranian dog with widespread eruption which we did not succeed in curing after 68 days' treatment, in which it was 12 times bathed, 9 times anointed all over with salve, besides receiving 9 separate local applications of the cantharides-ointment. We must add that a further 16 weeks of treatment with Peruvian balsam had no better resu't.

3. Inunctions with 1 per cent. *sublimate-ointment*, with washings of the same drug in a 1 per cent. solution, are also, according to our experience, a method that meets with success if long persisted in. But the dogs must be carefully hindered from licking off the salve, or they may easily be poisoned. This, therefore, is one of the most dangerous of remedies. Instead of sublimate ointment, Lemke recommends ½–1½ per cent. spirits of sublimate, or a sublimate vasogen ointment.

4. Rubbing with *creolin* in an equal quantity of spirit, combined with creolin-baths (2 per cent.), has also wrought a cure for us in a few not too advanced cases. According to Brandl and Gmeiner, the best remedy in acarus mange is liquor cresoli saponatus (lysol), which is very similar in its action to creolin.

5. Carbon bisulphide is recommended by Schindelka on account of its anti-parasitic and fat-dissolving properties. After careful washing, th· parts affected are covered for several minutes with pads of cotton-wool soaked in carbon bisulphide, which should be pressed well on to the skin. After the places have become quite dry, they should be covered with formalin paste (formaldehyde 1–3, vaseline 50, zinc oxyde and amylum āā 25). The treatment should be repeated every 3–4 days; after 3–5 applications there is usually a complete recovery.

Where the skin is very thick, Schindelka employs carbon bisulphide in combination with sulphur and neutral soap. The drawbacks to sulphur as a remedy for mange beyond its evil smell, are that its application causes smarting, and its inflammability.

These remedies may either be applied singly or one after the other, but must always be used with care and perseverance. The rubbings must be daily and should be accompanied by careful pressing out of all newly-formed pustules, the hair should be clipped from time to time, and the dog well fed. None of the other remedies mentioned above seem to be reliable in any but slight attacks. Better, perhaps, may be the *baths of sulphuret of potash* recommended by Mégnin, which he used daily for two months for fifteen minutes at a time. The sulphuretted hydrogen hereby developed is a deadly poison for the mites, which explains largely its favourable result, as it does also in Brusasco's process. Roth recommends the operative treatment of acarus mange (removal with a razor of the layer of skin containing the mites), with subsequent disinfection (powdering with xeroform). Eber confirms the good results obtained by this method.

Prophylaxis consists in separating sick from healthy dogs and in thorough disinfection of their kennels. This is a disease which ought to be legally proscribed. Sick dogs should be subjected to strict police inspection, and the incurable cases should be killed at once. The condition is so difficult of treatment that it can never be otherwise extirpated. Schindelka holds this view also.

SKIN DISEASES PRODUCED BY FILARIA.

1. Among *horses and asses* we find a peculiar, nodular skin affection which is caused by round worms (filaria hæmorrhagica s. multipapillosa), and is known as dermatitis pruriginosa (Rivolta) and dermatitis granulosa (Laulanié). According to observations made by the latter, the skin is in this disease permeated by numerous nodules of spherical or elliptical shape, which penetrate to its subcutaneous cellular tissue. The centre of each is soft, caseous and contains a round, spirally-coiled worm with characteristic transverse striation, which in the older nodules may be already decomposed. The skin itself is thickened. The centre of the nodule has plainly lost its capsule and can be peeled off; the process begins with small-

cellular infiltration of the cutis and subcutis. Laulanié believes that the parasite can remain for years in the nodule, and has found that the nodular eruption retrogrades in winter and develops afresh in summer owing to the freer flow of blood to the skin. According to Railliet and Moussu, the filaria inhabit the subcutaneous, intra-muscular and intra-fascicular connective tissue and penetrate from the subcutis to the skin. When the nodules burst they retire more deeply in. Contamine and Drouilly observed bleeding of the skin and hæmorrhagic skin-nodules in Hungarian horses, which were produced by the incursion of filaria hæmorrhagica, 2¼ to 2¾ inches long. Rivolta considers that the removal of the affected tissue is necessary for a cure. It is probable that many of the so-called "tubercular eruptions" described by earlier authorities, and especially by Haubner, belong to the chapter of granular dermatitis produced by nematodes.

2. In *dogs* Siedamgrotzky and Schneider observed a pustular eruption on the outer surfaces of both fore and hind legs which was caused by round worms. The small, intensely itching pustules and nodules, surrounded by a reddened area, were situated upon hairless parts and each contained one to three round-worm embryos, with a bodkin-shaped tail and from $\frac{1}{600}$ to $\frac{1}{36}$ inch in length, which had evidently wandered from the hair-sacs. They were to be found among the straw bedding along with full-grown ones $\frac{1}{25}$ inch long. Recovery followed without medical treatment within three weeks. Rivolta, Rosso, Mazzanti and others also found filaria in ulcerative skin eruptions of dogs.

Filaria medinensis.—The medina-filaria found in Egypt, India and other southern lands produce round worms, which grow to be $\frac{1}{8}$ inch in diameter and usually live as parasites in the subcutis of man and the domestic animals, where they cause local swellings and abscesses in the extremities. They are most common on cattle, dogs and horses. Where, as on man, generally only one filaria is found, we may meet with two, three, four and even five on the domestic animals.

SKIN DISEASES CAUSED BY FLIES, GNATS, GAD-FLIES AND THEIR LARVÆ.

General Notes.—Our domestic animals are plagued by the above-named insects in many ways. Some cause no real skin affection, but merely irritate the animals, prevent them from feeding and occasion waste of fodder. Among such are the

common house-fly (*musca domestica*) and, as a sweat-sucking fly, the raven-fly (*musca corvina*). Another section settle temporarily on domestic animals and suck blood, especially on tenderer parts of their skin. Such are the gad-fly of cattle (*tabanus bovinus*), the rain gad-fly (*tabanus s. hæmatopota pluvialis*), the blind gad-fly (*tabanus s. chrysops cæcutiens*), mosquitos, tsetse flies, etc. Others settle upon the animals in great numbers, and by their many stings cause serious inflammation. Such are the common stinging gnat (*culex pipiens*), the common stinging fly (*stomoxys calcitrans*), the storm-fly (*anthomyia meteorica*), the common and ornamented crawling gnat (*simulia reptans and ornata*) and, finally, the most dangerous of all gnats, the Columbacz gnat (*simulia Columbaczensis*). Many insects require also to be considered because their larvæ (maggots, mites) cause trouble on, in or even under the skin. Thus the blue blow-fly (*musca s. Calliphora vomitoria*) and the carrion-fly (*musca cadaverina*) lay their eggs in wounds and ulcers, as does also the grey meat-fly (*sarcophaga carnaria*). To this category belongs finally the most important of all, clinically considered, viz., the gad-fly (*œstrus bovis*), whose larvæ ar· found in the subcutaneous connective tissue. Among the above those of chief pathological interest are :—

1. The **Columbacz gnat** (*simulia Columbaczensis*, or *maculata*), an ash-grey insect about the size of a flea, whose ravages were first noticed in the neighbourhood of the Castle of Columbacz, near Temesvar, in Hungary, where in 1783 nearly 600 animals (horses, cattle, pigs, sheep) died from its stings. It is also found in Austria, Bohemia, Prussia and in scattered spots all over Germany. The insects breed in swarms after floods, forming thick blackish-grey clouds, which settle down on grazing animals in the open, so that white cows appear quite black. The animals become frantically restless, rush off home, fling themselves on the ground, groan and bellow. On examination the gnats are found, not only on the hair, but buried in all the natural openings of the body, and, as a result of their stings, painful swellings arise all over the body, and especially on the mucous membranes of the nose, mouth, eyes and vagina. The animals may perish in a few hours from suffocation produced by the swelling-up of their breathing passages. Those less severely attacked are ill for several days, eat little or nothing, and show the next day a number of small, hard, painful nodules on the skin, which do not pass away for 3 to 4 weeks, and also various

serious swellings of the mucous membranes. These inflammatory signs often remain for fourteen days or longer. On *post-mortem* examination of the fallen animals, we find intense, gelatinous and bloody infiltration of the subcutis, swelling and inflammation of the visible mucous membranes, as well as signs of hyperæmia and œdema of the lungs. Occasionally the insects have penetrated to the throat and larynx, or even into the wind-pipe, setting up inflammation and swelling there and even stopping up the lumen of the trachea. *Treatment* of the sick animals is restricted to cold washings and douches and scraping the swollen membranes until they bleed. Eventually tracheotomy has to be performed. As a *prophylaxis* washings with tobacco-lye are recommended; but all the fly-remedies may be tried, such as creolin, carbolic acid, tar, naphthalin, petroleum, fœtid animal oil, asafœtida, decoctions of walnut-leaves in vinegar, etc.

Simulia ornata.—A similar case of wholesale attack on cattle, horses, and sheep, caused by simulia ornata, is reported by Müller, when, out of 170 cattle attacked, 26 died.

Simulia reptans.—Bergmann states that in a Swedish province cattle, horses and sheep fall sick in hot summer weather as a result of the bites of the simulia reptans. In 1892, 15 head of cattle perished, one animal dying within the space of half an hour. The autopsy showed subcutaneous œdema, œdema of the lungs and plasmic effusion of the pericardium. Creolin mixed with olive or whale oil was recommended as a prophylactic.

2. The **skin gad-fly** (*œstrus sive hypoderma bovis* and *hypoderma lineata*) produces what are known as gad-fly boils (warbles), which are most troublesome in cattle, but are rarer in horses, asses and sheep, affecting chiefly the shoulders, loins, flanks and breast. The boils are caused by the female gad-fly laying her eggs upon the ox's skin, where they hatch, and the larvæ bore through the entire thickness of the skin and settle in the subcutaneous cellular tissue, where their further development takes place. This lasts about nine months (July to March). They produce inflammation and suppuration round about them and finally a tumour as large as a pigeon's egg (warbles). When the larvæ are ripe, they quit their lurking places after first widening the entrances with their hinder parts. The number of such boils on the same animal is usually small, but there may at times be 50 or even 100 on the same skin, producing a very injurious effect upon the animal, which rapidly loses flesh. On the approach of the flies the cattle begin "gadding," grow

restless, spring wildly hither and thither, bellow loudly, elevate their tails and seek if possible a refuge in water or in their byres. The *treatment* for warbles consists in squeezing out the riper larvæ, or opening them with a knife; and *prophylaxis* in giving attention to the skin, removing the eggs and rubbing the upper portions of the body with creolin, tar, dilute carbolic acid, solution of asafœtida or a decoction of walnut-leaves. Do this especially on sultry days which threaten thunder, and before sending the animals out to graze. It is also recommended to rub their backs with salt or rancid butter during the fly-season. The damage to the hides of cattle in Ireland by œstrus larvæ is estimated at ten million marks (nearly £500,000) annually.

Hinrichsen believes that he has found the larvæ of the gad-fly in the form of fine parasites in the canal of the spinal cord in cattle. It is his opinion that the cattle take up the gad-fly eggs with the herbage, and that the young larvæ penetrate from the stomach and intestines to the spinal canal, dwell there for five to six months and then migrate through the inter-vertebral foramina as far as just below the skin. According to Hinrichsen, these larvæ are to be found in the spinal canal of from 40 to 50 per cent. of all cattle that have been out at pasture especially during the months December to March. Ruser, Berg, Neumann and Horne also confirm the frequent presence of larvæ in the spinal canal. Ruser has succeeded in proving that the œstrus larvæ enter the system through the mucous membranes of the œsophagus. They bore through the œsophagal wall and enter the mediastinal tissue under the vertebral column. Thence they find their way into the vessels and nerves until they reach the sub-cutis or the spinal cord. This view is confirmed by Koorevaar, who further shows that the larvæ from eggs deposited in June are found in the œsophagus from July to September; they are present in the spinal cord from September to January; and in the sub-cutis from January to May.

A skin disease similar to gad-fly boils has been observed by Railliet in dogs, but caused by the larvæ of ochromyia anthropophaga (possibly larvæ of the human gad-fly, dermatobia noxialis ?) penetrating beneath the skin. Its symptoms were hard nodules, the size of a lentil and painful when touched, with a cylindrical opening in the middle. After six or seven days the nodules grow softer and enlarge to the size of a small nut, when the larvæ emerge. These nodules may occur anywhere, but seem to prefer the tail, ears and paws. Here, too, removal of the larvæ is the only cure.

Gad-fly boils in Horses.—The larvæ of flies are found singly in the skin of horses (hypoderma equi). Hell repeatedly observed gad-fly boils in horses imported from England, and in two cases saw considerable purulent inflammation of the sores resulting from saddle pressure. As in the case of the ox, the boils are generally found in the skin of the back, on the withers, upon the buttocks, and, occasionally, upon the neck and belly. They always, however, occur singly; never, as in the case of the ox, in patches.

3. The so-called **fly-larvæ sickness** of *lambs* is known especially in Holland, and is caused by the maggots of *musca Cæsar* (*lucilia Cæsar, L. sericata, L. macellaria*). The flies lay their eggs between the hind legs of lambs suffering from diarrhœa. The larvæ are said to seek out the more woolly parts of the tail root and loins, where they vegetate in clusters under the wool, perforating the skin like a sieve. Older animals are not often attacked. *Treatment* consists in removal of the maggots and application of carbolic solution, decoction of tobacco, etc. Certain blue-bottle flies seem to be of special interest in tropical countries. According to the reports of Frantzius (Virchow's "Archives," vol. 43), thousands of calves perish there owing to the maggots laid in their navels and the consequent local inflammation.

The remedies which may be applied to the skin as a protection against winged insects are: rubbings with highly odorous fluids, such as creolin (2 per cent.), sanatol, naphthaline, asafœtida, benzine, petroleum, stinking animal-oil, laurel-oil, oil of sassafras, oil of cloves, oil of eucalyptus, and decoctions of tobacco and walnut-leaves; also with bitter solutions such as decoctions of absinthe, gentian, nux-vomica, tanacetum, or datura stramonium. The alcoholic extract of Persian insect powder is also said to be very good.

LICE (HÆMATOPINUS) AND TRICHODECTES.

1. **Lice** (*Hæmatopinus*) are blood-sucking parasites, which attach their eggs (nits) to the hairs of the animals they infest. Most domestic animals have their special species, thus the *horse* has the hæmatopinus or pediculus equi, or macrocephalus, the *ox* has its hæmatopinus eurysternus and tenuirostris, the *pig* its hæmatopinus suis or urius, the *dog* is infested by hæmatopinus piliferus, the *goat* by hæmatopinus stenopsis and the *rabbit* by hæmatopinus ventricosus. The places most favoured by these parasites are the neck and back, also the root of the tail, in cattle the vicinity of the horns, and in pigs the inner sides of the hind thighs. Lice are most common on cattle, and are found even on the best nourished animals; the same may be said also of dogs. In general they thrive best on poor and ill-fed animals, and their presence is always an indication of a neglected skin. The phenomena produced by lice are itching, loss of hair, marked desquamation of epidermis and also numerous nits or eggs attached to the hairs. The animals often diffuse an unpleasant smell, and are (especially dogs) covered all over with a greasy or impetiginous matter.

2. **Trichodectes** are not blood-suckers, but *epidermis-eaters*. In contrast to the pointed head of the hæmatopinus, theirs is broad and shield-shaped; they live on epidermal-scales and hair, and their favourite haunts are the head, neck and legs of domestic animals. We distinguish the following varieties of the parasite: on the *dog* trichodectes latus, on the *horse* T. pilosus, on the *ox* T. scalaris, on the *cat* T. subrostratus, on the *goat* T. climax, and on the *sheep* T. sphærocephalus. The latter causes very intense itching in sheep, which may arouse suspicions of scab.

The following *drugs* are highly spoken of as remedies:— 1. *Grey mercurial ointment*, which may be applied in small doses on all domestic animals, especially horses, pigs and dogs. Even on full-grown cattle, which are known to be very sensitive to mercury-ointment, we have had capital results from rubbing about four scruples of the grey salve between the horns, without noticing any poisonous effect. For dogs it is best to smear the ointment under the collar in quantities about as large as a pea or a bean. We have also obtained excellent results in dogs from carefully washing them with one per cent. sublimate solution, doing only a small part at once and being particular to rub it quite dry afterwards. 2. *Persian insect powder*, which should be sprinkled between the previously moistened hair, is not strong enough for the larger animals. 3. *Tobacco-decoctions*, 4 to 5 per cent., are very good for sheep and dogs, but less to be recommended for horses and cattle; nicotina, a kind of tobacco-lye, is also well spoken of. 4. *Creolin* is especially commended by Gross for lice on horses. He applies it in a five per cent. solution with brushes, repeating the dose in six days and again in another six. Each such washing must be accompanied by a careful cleansing of the stable. 5. A mixture of petroleum and either linseed or colza oil, well rubbed into the affected parts, is recommended by Siebert, Eichhorn and others on account of its rapid action and freedom from dangerous sequelæ. Among the many other remedies we may enumerate lysol, bacillol, naphthaline, benzine, staphisgriæ in vinegar (1 : 20); a mixture of powdered cevadilla-seed, staphisagriæ and white hellebore in equal parts with two parts of aniseed; this, however, is only suitable for the larger animals; benzol, diluted with green soap, spirit or oil; carbolic acid in a five per cent. solution; and also for well bred dogs etherial oil of aniseed, mixed with ordinary oil or dissolved in spirits (1 : 10). Arsenic lotions (arsenic and potash, each ½ oz., water and vinegar each 3 pints),

as recommended by Viborg and Schleg. must be used with caution, but are especially efficient on large animals with many lice.

To destroy their eggs washings of vinegar may be applied (unless one prefers to await their development); this destroys them by dissolving their calcareous shells. Träger suggests that the lice should be combed off into a zinc bath containing lysol solution. Finally, and in obstinate cases, we recommend clipping the hair and disinfecting the stables. It is the rule in army stables to go over all the horses periodically, especially in the summer; the fetlocks, mane and tail are examined and the saddle-cloths disinfected.

VARIOUS OTHER SKIN-PARASITES.

1. **Fleas** occur on dogs (*pulex canis*), cats (*pulex felis*), and on rabbits (*pulex goniocephalus*). They are blood-suckers, which not only irritate their hosts with great itching, but also give rise to chronic skin-eruptions. The skin is, moreover, soiled by the dung of the fleas, which is often found thickly scattered among the hair like coffee-grounds. The best remedy for fleas is the Persian insect-powder (*flores pyrethri*), which should be dusted over the skin, after previously slightly damping it in order that it may adhere better. It is also advisable to let the dog remain out of doors for a time after sprinkling, lest the cast off fleas find a lodgment in the stable or house. Also washing dogs with a two per cent. solution of creolin is a very effective cure. A very troublesome parasite of domestic animals in America and Africa is the sand-flea (*sarcopsylla penetrans*).

2. **Ticks** are large insects resembling mites or acari, which live in woods, bushes and grass, whence they attack animals and bore into their skin in order to suck blood. We distinguish the dog-tick (*ixodes ricinus*), which is found on cattle and sheep as well as on dogs; and the ox-tick (*ixodes reticulatus*), which settles as a parasite upon the skin of cattle and sheep. The American tick (*ixodes Americanus*) is said especially to attack horses. Junach found 30–300 ticks upon each of the eight horses of a patrol which had been standing for an hour among bramble bushes. Joly observed a nodulous exanthema in saddle-horses, due to tick-larvæ. According to Janson, ticks are a positive danger to horses in Japan, where many succumb yearly to their attacks. Salles saw cattle with ulcers and accumulations of pus as large as a hen's egg on the back of the neck.

Respecting the cattle-ticks (*boophilus bovis*, *ixodes annulatus*), which stand etiologically related to Texas fever, compare the chapter on "Texas Fever" in Vol. II. *Treatment* consists in the application of common fatty oil, creolin-oil, carbolic oil, or of the etherial oils (turpentine, aniseed, caraway and fennel oils), or of tobacco-decoctions, petroleum, etc. These should be put on to the tick as found with its head inserted into the skin. It is not well to pluck it off violently, as the head then remains behind in the skin.

3. **Louse-flies** (*pupipara*) occur as parasites on horses (*hippobosca equina*) and upon sheep (*melophagus ovinus*, sheep ticks). The louse-fly of horses settles about the tail and anus and upon the lower part of the belly, and excites chiefly by its rapid running about rather than by biting or sucking, rendering the animal very restless. The louse-fly of sheep occurs chiefly upon grazing sheep, of whom it sucks the blood. It excites intense itching, which makes the animal rub itself and damage its wool. The excrements of these flies often dye the wool green. *Remedies* for sheep are tobacco-decoctions, washings with solutions of creolin or of carbolic acid, as well as grey mercury-ointment, 2 to 3 scr. each rubbed along the back and under the neck, and all such treatment is most efficacious after shearing.*

4. The **bird-mite** (*dermanyssus avium*) infects especially fowls, pigeons, swallows and cage-birds, as well as their roosts, nests and cages. The colour of these mites is blood-red or reddish-brown, and at night they betake themselves to any animals that may be near (cattle, horses, dogs and cats), upon whom they may permanently settle, causing much itching and an eruption of the skin. Thus Steinbach noticed a mange-like eruption on horses, with isolated scabs and fissures and accompanied by intense itching. The mites were found in the fore-lock and mane and at the root of the tail. Möbius also saw a similar skin affection on eight cows, with round hairless patches, which was vainly treated for two years, and only disappeared on the removal of certain swallows' nests in which the bird-mites had dwelt. Prietsch found a great number of sore spots on two horses, viz., on their necks and backs, which were devoid of hair, partly covered with bran-like scales, and partly

* *Hippobosca equina* is common in the New Forest. In horses new to the district it often causes dangerous excitement. Kicking and bolting have frequently resulted.

with thin scurf. The itching was considerable, especially towards evening and in the night. According to Trasbôt, these mites prefer to settle on horses underneath their cloth cover. Moreover, these bird-mites, by settling in the outer ear-passages of cattle, may produce an otitis externa (Gassner) and, by penetrating to the middle ear, give rise to peculiar signs of cerebral irritation. Thus Stadler and Schuemacher noted in cattle slaughtered on account of mania that the inner parts of the ear were quite choked with these mites. (Compare also the skin diseases of birds.) In the ear-passage of a cow which used to shake its head for hours together, Ostertag found an allied form of mite, viz., gamasus auris. *Treatment* consists in removing the hen-roosts, swallows' nests, etc., from the neighbourhood of the cattle, and in the application of mite-killing remedies (creolin, carbolic acid, etc.). A thorough examination and cleansing of the byres is also advisable.

5. The **autumn grass-mite** (*leptus autumnalis*) lives in dry grass, among the ripe corn, and also on lilac and currant bushes, whence it travels to the backs of horses and dogs, causing a pustular skin-eruption, chiefly on the head, round the mouth, ears and eyelids, and also upon the inner sides of the hind legs and on the genitals. The local changes are at first circumscribed red spots, from a poppy-seed to a hemp-seed in size, which on closer examination are found to be papules or pustules, on which rest a number of the red-coloured mites. On pressure, a bloody fluid can easily be squeezed out of them. Later these spots enlarge to the size of a shilling, are bare or but sparsely haired, the skin becoming hyperæmic and sensitive, and the hair falling out. There is usually also a moderately severe itch. This eruption may last for several weeks, but can easily be removed by rubbing with creolin or carbolic ointment, or with creolin or carbolic glycerine. Eloire has also found the mite on fowls. According to Mégnin, the autumn grass-mite forms the embryo of the velvet-mite (the six-legged larva), *trombidium holosericeum.* It is supposed to cause the vesicular eczema of the grape-sickness, and also to give rise to other skin eruptions, accompanied by severe itching, in dogs, cattle and horses. It is said to be also met with on the leaves of buckwheat. According to Zürn, the mites are carried into the house by dogs and conveyed in this way to man.

6. **Bed-bugs** (*cimex lectularius*) sometimes attack dogs during the night, or they may infest the baskets of puppies.

They cause an eruption which takes the form of hard nodules, slightly pigmented, with visible puncture-marks (Schindelka).

APPENDIX.

SKIN DISEASES OF POULTRY PRODUCED BY ANIMAL PARASITES: THE MANGE OF BIRDS.

Among the multitude of animal ecto-parasites which infest our domestic poultry, the mange-mites play a principal part, and may be divided into two chief kinds :—

1. *Dermatoryctes mutans* (Ehlers), a mite resembling sarcoptes, which was formerly called sarcoptes mutans (Robin), sarcoptes avium (Gerlach), or cnemidocoptes viviparus (Fürstenberg).

2. A mite discovered by Caparini (*dermatophagus gallinorum*), which produces a dermatophagus or symbiotic mange. According to Troussart and Neumann, this mite is identical with epidermoptes bilobatus and bifurcatus (Rivolta).

1. **Dermatoryctes-mange** is caused by a mite very like the sarcoptes-mite of mammalia, but not identical therewith, and which is distinguished from a sarcopt by the structure of its cutting jaws. The females are larger than the males (0.4 mm. long) and are almost spherical in shape. The males are more oval (0.2 mm. long). These dirty-yellow mites possess four pairs of feet when mature and only three when in the larval state; on the anus grow two long hairs. The dermatoryctes mite, as its name indicates, is a burrowing insect, which bores tunnels under the epidermis. It was discovered in 1859 by Robin and Lanquetin. Fowls are most subject to this mange, and imported breeds more so than native ones. The mite can pass to other birds, such as pigeons or exotic birds, and Mégnin has found it on turkeys, pheasants and singing birds. Ostertag has observed its passage to a horse. Under the name of sarcoptes lævis var. columbæ, Railliet first described in 1885 a mange-mite in pigeons which bears a strong resemblance to dermatoryctes mutans. Writing again later (1887), he describes a second variety (sarcoptes lævis var. gallinæ), which he discovered in the mange of poultry. The type is subject to considerable variation, and it is certain that the varieties described by Railliet are identical with the dermatoryctes found by Friedberger in pigeons in 1886–7. According to Railliet this mange is

only slightly contagious. According to Brandl and Gmenier, the dermatoryctes mite remains active for 12–14 days after separation from its host.

The *symptoms* of this gallinaceous disease, which is also known in Germany as "chalky legs," and as the "fish-scale sickness" (ichthyosis) of fowls, are as follows:—The mange settles mainly on the legs, but may obviously be transferred by the bird's scratching and pecking to the head (comb, wattles, beak). At first we notice upon the front surface of the extremities (corresponding to the metatarsal and adjoining bones) small, whitish-grey spots, which enlarge and grow to round or zig-zag crusts. Both the front and back surfaces are later covered with uneven, lumpy, yellowish-grey scabs, which are very porous and cracked, may become $\frac{3}{8}$ inch thick, and are of foliated structure, some of the leaves having a greasy polish like mother-of-pearl, and feeling greasy to the touch. By this scabby incrustation the legs are thickened to deformity, the horny foot-scales become detached and stand out, and the skin appears as though coated with chalk or lime (so-called chalky leg). The scabs consist mainly of epidermal-scales, dry products of inflammation and mites, beneath which the skin is red, swollen, purulently infiltrated and easily bleeds. There is usually moderately severe itching, and the birds peck at the scabs and constantly rub their legs. In severe forms of the disease they cannot run and can scarcely stand, but squat upon their breasts. Later emaciation and cachexia show themselves, and in very severe cases death even may result.

The *treatment* consists in removing the scabs after a preliminary softening with glycerine or soft-soap, and in rubbing with ointment of creolin, carbol or creosote (1 : 10 to 20), tar, Peruvian balsam, styrax, Helmerich's sulphur-salve, etc. Brandl and Gmeiner particularly recommend lysol (identical with liquor cresoli saponatus). At the same time sheds and roosting-places must all be disinfected.

2. The **Symbiotic-mange** of poultry attacks especially the neck and breast, but may quickly spread all over, including the comb and wattles. The skin becomes covered with thin, transparent, straw-coloured epidermal-scales, which form in layers one on the other, and may grow to thick, dry, doughy scabs of a dirty yellow-grey colour. The skin beneath is hyperæmic, and there is no itching. This symbiotic-mange quickly leads to loss both of flesh and strength, and sometimes

to death. *Treatment* is the same as for the preceding form of mange. Friedberger noticed a complication of dermatoryctes and symbiotic-mange upon the same animal.

Under the name of **Sarcoptes cysticola** (*laminosceptos gallinarum s. cysticola, symplectoptes*, or connective-tissue mite) a mite resembling a sarcopt has been described by several writers. It is discovered enclosed in a capsule, in the form of a whitish or yellowish, oval nodule, in the skin, the subcutaneous cellular tissue, the inter-muscular connective tissue, as well as upon the serous membranes of the organs in the abdominal cavity. It sometimes produces a mange-like eruption upon the skin (Unterberger).

VARIOUS OTHER SKIN DISEASES OF POULTRY CAUSED BY ANIMAL PARASITES.

Of the various animal parasites which inhabit the skins of poultry, and cause inflammation there or injury of plumage, we may mention the following :—

1. The **demodex of the tame pigeon** (*harpirhynchus nidulans*) lives in the feather-sacs, which are thus expanded into yellow capsules or cysts as large as a pea or even a bean ; also in the connective tissue cysts of the skin, particularly on the lower breast and beneath the wings. Their knob-like protuberances consist almost entirely of mites, hundreds of which may be found in a single cyst. When many such cysts are present upon the skin the birds quickly grow thin and perish.

2. The **worm-shaped pigeon-mite** (*hypodectes columbarum*) is found in the subcutaneous cellular tissue, upon the serous membranes of the abdominal cavity, and also in the connective tissue of the aorta, and represents the larva or chrysalis (nympha) of falciger rostratus. It hinders the processes of moulting and feather production, and also causes slight inflammation.

3. The **Feather-quill Mite of Fowls and Pigeons** (*syringophilus bipectinatus*) lives in the quills of the tail and wing-feathers, and causes deformity in the quills, loss of feathers and irregular moulting. The feathers have a sickly, dull, lustreless appearance, and are usually bent at the point of their pivot ; the quills lose their transparency, and become filled with a dirty,

yellowish-grey powder. The mites do not seem to cause any further injury. Heller makes a statement that in Schleswig-Holstein from 70—90 per cent. of all fowls are infested with syringophilus bipectinatus. On the peacock we find syringophilus uncinatus. On the other hand, the mite analges minor, discovered by Nörner, which inhabits the interior of the quill, is said to be entirely harmless, and not to affect the feathers in any way, so that its interest is more scientific than practical.

4. The **Feather-mite of Pigeons** (Zürn) lives in great numbers among the feathers, and may give rise to emaciation and even fatal debility. Zürn presumes that the so-called scabies of pigeons on the front part of the head is caused by this mite. (Apparently it belongs to the mallophagi.)

5. The **Common Bird-mite** (*dermanyssus avium*) must be reckoned as a blood-sucking parasite, and one of the most troublesome and dangerous of all for poultry. It occurs mostly on fowls and pigeons and also on cage-birds, which it generally attacks at night. It also lodges on horses, dogs, cattle, cats, and even on man. Its abode during the daytime is in crevices of the floor, cracks in the walls or cages, and also on the under side of the perches. Occasionally, however, the mite changes its habits and appears to permanently infest its host, seeking those spots which are unexposed to light or to defensive measures. The unrest caused among poultry by these nightly visitors is considerable. Indeed, young ones may even perish from loss of blood, and the older ones grow anæmic and thin. Zürn found that the so-called "greedy sickness" or bulimia of canaries is due to the presence of enormous numbers of bird-mites. They are also often found in the nostrils, where they produce catarrh of the mucous membrane, from which younger birds may die; also in the auditory passages. Similarly, the mite holothyrus coccinella attacks ducks and geese in the East Indies.

6. The **Autumn Grass-mite** (*leptus autumnalis*) is also found on fowls. It bores into the skin at the root of the feathers, producing red dots and little pustules. In consequence of the intense irritation, epileptiform fits may supervene. Young s hypscream, flap their wings, fall to the ground and mostly die quickly le. days. Older ones perish of anæmia.

7. The **Tick of Pigeons** (*argas marginatus s. reflexus*) plagues the pigeons of France and Italy by sucking blood in the night, and may even kill them. It produces a toxic albumen, which has some resemblance to the poison of the viper and puff-adder. Sometimes it also bites men (Alt).

8. **Feather-lice** are wingless mallophagi (fur-eaters) and correspond to the hair lice of mammalia. They feed exclusively on feathers and epidermal-scales, but can attach themselves to the skin by their eating apparatus. For details of their families and species, see Zürn's "The Diseases of Domestic Poultry." In large numbers, these creatures can cause disturbance of nutrition in their hosts; and they multiply fastest on sick birds.

9. The **Bird-flea** (*pulex avium*) specially infests pigeons.

10. **Bugs** (*acanthia lectuaria*) may spread themselves from beds to poultry and pigeons (Lucet).

11. **Louse flies** (*ornithobia pallida* and *ornithomyia avicularia*) are occasionally found on hens. Wingless insects of the order collembola, species isotoma, if present in large numbers, occasionally injure the condition of fowls (Theobald).

12. Finally, the **larvæ of beetles** (*tenebrio molitor, dermestes lardarius, necrophorus, silva*) are said to attack pigeons, and sitting hens, and even inflict upon them fatal injuries (Kitt, Mégnin).

For the *extirpation of these numerous parasites* many remedies are recommended. According to Zürn, the etherial oils (aniseed, rosemary) diluted with 1 : 20—50 of water or oil are good against feather-quill mites and feather-mites (Nos. 3 and 4 above); the same, too, with Peruvian balsam and styrax (1 : 4 spiritus), combined with great cleanliness and an abundance of sand. Sprinkling the plumage with ½—1 per cent. creolin-water is a very excellent plan. Against the bird-mite (No. 5) we use Persian insect-powder, or blow sublimated sulphur on to the feathers, or wash them with dilute etherial oil of aniseed. Moreover, the cracks and crevices of the wood-work, perches, etc., must be smeared on a morning with creolin, carbolic acid or petroleum, and the shed if possible swilled with hot water, to which a small quantity of the etherial oil of aniseed has been added. When the fowl-house is of brick, its walls should often

be whitewashed, and bird-cages scalded. For feather-lice we also commend Persian insect-powder, or any etherial oil; also decoctions of aniseed, parsley-seed, etc. The birds should also have abundant opportunity to bathe themselves in sand, which enables them to get rid of the parasites. The sand is best mixed with ashes, which may with advantage be scattered alone upon the floor of the fowl-house. Finally, it is recommended that the roosting perches be isolated by oil-cups fixed upon the bearers which carry their ends. It is also said that by hanging a pocket-handkerchief over the bird-cage at dusk and removal of the same at daybreak the mites may be caught and destroyed.

CHAPTER XI.

DISEASES OF THE LOCOMOTORY ORGANS.

MUSCULAR RHEUMATISM.

Notes on Rheumatism in General.—Under the name of "Rheumatism" have long been classed all those diseases of the locomotory organs which arise from cold, and run their course causing severe pain in various parts of the body, and producing much incapacity for movement in the organs affected. Among these organs special mention must be made of the muscles, aponeuroses, tendons and tendon-sheaths, of the periosteum and also the articular synovial membranes. The connection of all these "*rheumatic*" affections is obviously doubtful; and we might with equal justice include all other complaints which arise from cold under the same name, such as rheumatic peritonitis, pleuritis or colic. An even stronger reason for rejecting this classification lies in the fact that, according to more recent investigations, cold cannot be accepted as the exclusive source of these "rheumatic" diseases of the locomotory organs, some of which are probably of infectious origin. It is, therefore, advisable to divide them, and to consider as rheumatic that form which is really caused by cold, viz., muscular rheumatism, leaving for separate treatment the articular form, an evidently infective condition. In our domestic animals a differentiation between muscular and articular rheumatism is not so easy as in man, and is in many cases impossible. The diagnosis of muscular rheumatism in particular is often rendered very difficult by the absence of all subjective symptoms; and, moreover, complications of the two forms are much commoner in domestic animals than in man. Besides the difference of etiological relation, the variation of course also demands a separate description. Muscular rheumatism must be regarded as muscular inflammation (myosotis rheumatica), whereas articular rheumatism

represents an inflammation of the joints (polyarthritis serosa). The two conditions have nothing in common beyond their accompanying motor disturbances and pain.

Etiology.—Muscular rheumatism, or rheumatic muscular inflammation, is most common among horses, dogs and cattle, but is not rare in sheep and pigs. Of the dogs treated in the Berlin dog-hospital, just over two per cent. suffer from this complaint Cold is universally regarded as its chief cause. In the first place, we put damp, cold weather, cold winds and draughts, then badly kept stables, wet pastures and rapid cooling .of the body after violent exertion, etc. For instance, in the breeding establishment at Mezőhegyes acute muscular rheumatism appears almost as an epidemic among the foals of from six months to two years old (Tatray) in spring and autumn, and as the result of cold. Cold baths are in sheep a cause of muscular rheumatism, as well as lying out of doors on very cold nights ; and in dogs a cold sleeping-place or fetching objects out of very chilly water ; consequently the disease is much more common in the colder seasons. Among very pampered or young animals we find a predisposition, and also among tender merino lambs, or animals that have long been kept in warm stables, or been too highly fed or too little exercised. For the latter reason the ailment is common among fat dogs. Animals once attacked are more liable to a recurrence of the trouble. Exactly how muscular rheumatism is caused by cold is not yet satisfactorily explained. Probably the irritation produced by the cold upon the temperature nerves of the skin is by them reflexly transmitted to the nerves and vessels of the muscles, causing first of all disturbances of circulation and changes in the composition of the individual muscular fibres, which finally assume the character of a decided muscular inflammation. This explanation is based upon the physiological fact that, even under normal conditions, the metabolism in the muscles is stimulated by the action of a lower temperature upon the skin.

Older Theories.—The earlier opinion that the effect of chill was to suppress perspiration and thereby throw back certain injurious substances into the blood, and in consequence of this blood-dyscrasia set up inflammatory changes in the blood, can no longer be accepted. Neither does the hypothesis of an increased generation of lactic acid in the muscles, as a connective link between the chill and the muscular affection, appear to be justified ; for neither by artificial suppression of the activity of the skin (varnish) nor by the introduction of lactic

acid into the body, has anyone yet been able to produce rheumatism. The conception also of muscular rheumatism as a purely neuro-pathological process or muscular neuralgia is contradicted by the proof of inflammatory changes in the muscles themselves. Nevertheless, it must be admitted that under the name and aspect of muscular rheumatism several other disturbances—neuralgic, traumatic and circulatory—have often been included. Evidently a muscular inflammation exactly resembling, clinically, a rheumatic myosotis can also be produced by over-exertion ; but such traumatic muscular inflammation must not be confounded with muscular rheumatism, although it is by no means always easy to differentiate between them. Leube has declared that muscular rheumatism is an infective malady, an opinion confirmed by Peltesohn.

Post-Mortem Conditions.—In mild cases we find no pathological change in the muscles attacked by rheumatism ; but this is only when the hyperæmia or slight inflammation present during life is imperceptible after death (like the disappearance after death of milder catarrhal affections upon mucous membranes). When the attack is more severe, the well-marked signs of muscular inflammation are all present ; hyperæmia, hæmorrhage, serous exudation into the interstitial connective tissue (perimysium internum), softening, discoloration and disintegration of the muscular fibres. Where the rheumatic condition is of long standing, it may ultimately give rise to inflammatory proliferation of the muscular connective tissue, leading to the formation of so-called rheumatic nodules, that is, deposits in the connective tissue. Considerable atrophy of the affected muscles may also occur. According to experiments made by Zschokke, the pathological changes in the muscles of the horse are cloudy swelling, fatty and hyaline degeneration of the muscular fibres, hyperæmia, and moderate infiltration of the small connective tissue cells, with tendency to rapid regeneration of the atrophied muscles. Siedamgrotzky found in all the aponeuroses of a dog a diffuse redness and serous infiltration, the muscular apparatus being bright red and the capillaries injected ; and in another dog, suffering from chronic rheumatism of the masticatory muscles, an intense interstitial chronic myosotis in all these muscles. Bruckmüller also saw the inter-muscular connective tissue infiltrated with serum and charged with a reddish yellow, gelatinous fluid, the muscular fibres, which were very pale, had lost their transverse striation and undergone fatty degeneration. Upon post-mortem examination of a horse which had suffered from severe muscular rheumatism, Schmid observed signs of *poly-myocitis hæmorrhagica.*

Symptoms.—A. **In Horses.** The phenomena of muscular rheumatism in horses are usually restricted to single groups of muscles, particularly those of the extremities; rarely does the condition involve the entire muscular apparatus of the body. The animal is attacked suddenly, displays a stiff, strained posture and action of the parts affected, viz., the extremities; can only with difficulty be made to stand up or move backwards, takes very short steps, keeping its joints rigid and straight as it does so. Such movements are often accompanied by a peculiar, loud cracking of the joints. The affected muscles are painful and hard to the touch; and sometimes their entire surroundings are in a state of œdematous swelling.

A characteristic sign is the shifting (metastasis) of the rheumatism from one part of the body to another, *e.g.*, from a fore to a hind leg, when the limb first attacked may very quickly recover. Also the marked liability to a recurrence of the disease, and its improvement or even complete disappearance after prolonged exercise are further characteristics. The muscles most frequently attacked are those of the shoulders (omodynia rheumatica) and of the loins (lumbago). When the shoulder is affected its power of movement is hindered, especially in raising and advancing the limb, the stride is shortened and the horse goes "shoulder-lame." On backing, the foot is dragged and lameness is increased on going uphill or on soft ground. When the extensor muscles are particularly affected, the lameness is marked on turning the animal quickly. Lumbago shows itself in a stiffer carriage, sensitiveness and weakness in the lumbar region, a dragging gait, difficulty in getting up, and even in complete immobility of the hind legs, so that the hind quarters sink and the horse cannot rise again. The muscles around the hip-joint may also be implicated (hip-lameness), when a dragging gait with diminished flexion and forward movement of the affected limb are observed, and even a collapse if more weight be thrown on that side than on the other. It is not easy to decide, in the absence of all subjective symptoms, whether rheumatism of the inter-costal muscles (pleurodynia rheumatica) ever occurs in horses as in men. With these local phenomena we observe some degree of fever when the muscular rheumatism is widespread (we have noted 104° Fahr.), also increased rapidity of pulse and respiration; but, on the contrary, when the rheumatism is local there is no fever, and only a slight increase in the pulse-beats. Severe cases of muscular rheumatism are sometimes complicated

with other disorders arising from chill, such as catarrh of the breathing and digestive apparatus, with colic, pleuritis, affections of the tendons and grease; and also, though rarely, with articular rheumatism. In this manner the aspect of the disease is naturally much altered and may even take a fatal turn.

The *course* is very often rapid, when it is marked by a sudden disappearance of the gravest symptoms and a somewhat brief duration of the whole illness. Recovery then occurs in a few days or, at most, a week. There is, however, always a tendency to relapse. Muscular rheumatism often takes a chronic course and may become extremely obstinate and drag on for weeks and months. This is especially the case when the shoulders or loins are attacked. In rare cases of severe and general rheumatism death may result.

B. **In Cattle** the symptoms of muscular rheumatism are similar to those in horses, only that complication with articular rheumatism is more frequent. The favourite seats are the shoulders and lumbar region. It may be localised or almost every muscle of the body may be implicated, so that the beasts stand quite stiffly, unable to stir either neck or limbs and groan piteously. Sometimes the appearances are like those of epizoötic cerebro-spinal meningitis. The affected muscles feel tense and painful to the touch. In very acute cases the internal temperature is raised (104–106° Fahr.), and the breathing is occasionally extremely laboured. Regarding the course of muscular rheumatism we find that in cattle it frequently becomes chronic; but severe acute attacks may rapidly lead to death.

C. **In Dogs** the rheumatic process is usually localised in the muscles of the neck, shoulders and back, but sometimes affects the whole body. A prominent symptom is the animal's yelping; many howl on the least attempt at movement or on the gentlest touch, others even from fear of being touched. It causes a dog the greatest pain to lift it from the ground, elevate its head and stretch out its limbs or attempt palpation of the psoas muscles (lumbago rheumatica). The animal's movements become stiff, strained, timid, its neck is often held with peculiar stiffness, and the patient is frequently quite unable to move forwards, but lies down continuously. The ascent of stairs is particularly difficult and often impossible. But the sensitiveness of the separate muscular groups becomes very

quickly modified, and those which have seemed most severely crippled may often in a very short time be fit for use again, while the rheumatic condition has meanwhile settled elsewhere. The dog's appetite is often quite normal even during very severe attacks, but mastication is naturally impeded if the jaw muscles be among those attacked. Constipation is also often observed, as the attitude for evacuation has become painful and is therefore avoided when possible. Muscular rheumatism in dogs occasionally takes a chronic course, and these inveterate cases sometimes defy the most persevering treatment for months, when muscular atrophy may develop.

D. **Among Sheep** it is usually lambs which are attacked by muscular rheumatism, and the condition may be either local or general. The animals walk with a stiff gait, as though upon stilts, hold both back and neck rigidly, the head and neck are often persistently bent to one side, and there is frequently lumbago. The affected muscles are painful and hard to the touch; movement is avoided and the lambs remain lying. The course is generally rapid, and cure may take place in from four to six days, but in other cases the disease becomes chronic.

E. **In Pigs**, as in cattle, this form of rheumatism is often complicated with the articular variety. The animals have stiff legs and walk stiffly and with pain; the back also is stiff. Gastric disturbances are also said to be an accompaniment in pigs, and a weakness of the hind parts resembling paralysis often results. An epizoötic occurrence of muscular rheumatism was observed in Hungary among yearling pigs.

Differential Diagnosis.—Muscular rheumatism may be confounded with various surgical affections of the extremities, particularly those of the hoofs, tendons, bones and joints. (See surgical text-books on this subject.) It can be easily distinguished from articular rheumatism, which is always accompanied by swelling, raised temperature and painfulness of the joints; though in articular rheumatism the inflammation of a deep-seated joint which cannot be reached for palpation may always present the aspect of muscular rheumatism; when we must endeavour to find out if any of the muscles be affected and painful to manipulation. The differentiation of rheumatic lumbago from paralysis of the hind-quarters caused by spinal affections or external traumatic influences is more difficult

In such cases the cause and other symptoms of muscular rheumatism are most important. Rheumatic lumbago in horses, as seen in the course of acute muscular rheumatism, greatly resembles the phenomena of the paralysis of the hind - quarters which appears in hæmoglobinæmia, and must also be regarded as a rheumatic myosotis. In ordinary muscular rheumatism, however, we have no hæmoglobinæmia or hæmoglobinuria. Rheumatic hæmoglobinæmia and muscular rheumatism in horses are, therefore, ailments which differ only in their intensity. Muscular rheumatism may be confused with pleuritis, nephritis, osteo-myelitis and certain rheumatoid affection, such, for instance, as appear in the course of many infectious diseases, say, in cases of bronchi ectases. In cattle the malady is sometimes mistaken for cerebro-spinal meningitis.

Therapeutics.—The treatment of localised muscular rheumatism is chiefly external, and consists in rubbing the affected parts, after previously sprinkling with irritant remedies, *e.g.*, spirits of camphor, either alone or with oil of turpentine or of rosemary (1 : 10), etherial mustard oil (1 : 50), spirits of ammonia (1 : 15), tincture of arnica, etc. In this operation, friction and not the drug plays the chief part; we may also use Priessnitz's bandages, or cold douches, followed by warm packing, or massage, and, finally, try to excite local perspiration. To accomplish this the affected parts should be washed with soapsuds as warm as possible, vigorously rubbed and then warmly wrapped up. In chronic and very obstinate rheumatism of the shoulder, a subcutaneous injection of veratrine is highly recommended. For a horse we inject it beneath the skin of the shoulder in doses of ¾–1½ grains in 15–30 minims of alcohol, increasing the dose daily by about one-sixth of a grain, and omitting the injection every fourth or fifth day. After each operation it is well to exercise the horse, owing to the signs of restlessness which immediately follow it. Painful affection of the muscles of the loin (lumbago) in dogs may be relieved for a time by the injection of morphia. But the combined use of morphia and atropine in rheumatism of the shoulder in the horse is contra-indicated, on account of the dangerous by-effects (colic, symptoms of poisoning) which it provokes.

In cases of general muscular rheumatism the internal administration of salicylic acid or salicylate of soda is most to be commended. The internal administration of the salicylates may also supplement the purely local treatment in cases of local

rheumatism. To larger animals (horse, ox) we give both remedies in doses of $\frac{3}{4}$–1$\frac{1}{2}$ *oz.* three times a day, to smaller animals (dog, pig, sheep) 30 to 120 grains a day. As a rule, salicylate of soda is to be preferred to the acid, which, having a slightly corrosive action on the mucous membranes, is apt to interfere with appetite. This salt is usually given to large animals in pills or electuaries, to smaller ones in solution. The following formula is suitable for horses: Rp. Sodæ salicyli. 3$\frac{1}{2}$ oz., Pulv. Rad. Liquirit. $\frac{7}{8}$ oz., Farin secae. et aqu. fontan. q. s. f. Electuar. M. D. S., a third part to be given every three hours. Dogs and swine should be prescribed for according to age and size: Rp. Sodæ. salicylici, 31 grs.–2 drs., Aqua. destill. 6 oz. M. f. sol. D. S. A tablespoonful every hour. An ounce may be given to the horse in the form of an electuary every three hours until three or four doses have been given. In the case of the dog the solution may be repeated hourly until acute symptoms have disappeared. Salol is also used in place of the two remedies just named, and is said to be preferable to salicylic acid, as it leaves no bad after-effects and does not irritate the stomach; it seems moreover to act in the body like disguised salicylic acid. Administer salol to dogs in doses of 4–16 grains and up to 16 grains to 1 dram per diem, but for the larger domestic animals it is generally too expensive. We can confirm its good effects from personal experience. Antipyrine is also a good remedy for rheumatism in dogs, and we prescribe it in the following form: Rx. antipyrin, 1$\frac{1}{4}$ drams; aq. destill., 5 oz., a tablespoonful every three hours. Salipyrine, antifebrine and salophen have a similar effect.

The administration of laxatives is also very common, and tartar emetic especially is given to horses in feverish, acute muscular rheumatism. For dogs tincture of colchicum has been recommended as a gentle purgative and rheumatic specific —5 to 15 drops twice or thrice daily. A general perspiration may also be obtained partly by external means (wrapping the whole body in wet clothes and then surrounding it with woollen rugs), and partly by diaphoretics internally administered. Of these, the only ones which act upon horses are arecoline hydrobromide (1$\frac{1}{2}$ grains) and pilocarpine hydrochloride in very large doses (full-grown horses 6–12 grains; foals 3–5 grains, dissolved in 1–2$\frac{1}{2}$ drams of water), and injected beneath the skin. Hübner thus used pilocarpine successfully on a nine-months old foal against muscular rheumatism (4 grains in 1 dram of water). Sweating did not break out for two hours, but lasted

then for three hours. Siedamgrotzky, on the contrary, had no success with three horses after 4–5 days' continued injection of 3–6 grains of pilocarpine. Of course, all chill must be carefully warded off from animals thus freely sweating. They must also have rest, be kept warm and be moderately dieted.

Acute Muscular Degeneration.—An acute parenchymatous muscular degeneration, with symptoms of parenchymatous myositis (swelling, pain), makes its appearance after unwonted muscular exertion. It is seen in horses as a result of over-strain; after long rides; on racecourses (so-called "*racing sickness*"); after being tied up for some time with too short a head-rope. It is observed in cattle after long standing upright on railway journeys (so-called "railway-sickness"); after severe draught work; after parturition; and in severe cases of hæmoglobinæmia. The muscles most frequently affected are those of the back and loins, and the psoas, quadriceps and anconeus muscles. According to the degree, extent and localisation of the muscular degeneration, there is either a general paralysis or a paralysis of the loins. Or there may be only a weakness in the sacral region, or the paralysis may be confined to the patella extensors or the elbow extensors. Occasionally symptoms of colic (so-called "fatigue" colic) and high fever are observed. The principal macroscopic change lies in the pale, light-grey appearance of the muscles, which frequently resemble fish-flesh in colour, and look as if boiled. They are also abnormally brittle. Occasionally there are hæmorrhages into the muscular tissue. Under the microscope, signs of secondary inflammation and of various forms of muscular degeneration are to be seen, *i.e.*, cloudy swelling (albuminoid), fatty and hyaline (waxy) degeneration. The disease terminates variously; sometimes the animals die within a comparatively short time as the result of general paralysis; in other cases, degenerative muscular atrophy of the affected parts develops (which may be either acute or chronic), with permanent weakness and symptoms of paralysis. The treatment consists in slinging, massage, electricity, and the sub-cutaneous injection of caffeine and camphor.

ARTICULAR RHEUMATISM.

Definition.—Articular rheumatism is an infective febrile disease accompanied by inflammation of several joints and general systemic disturbance. It is more common among cattle than other animals, but is found in goats, dogs and pigs; rarely in horses. Out of 70,000 dogs treated in Berlin, there were only 92 cases of articular as against 1,462 of muscular rheumatism. Among 8,000 horses treated at the Berlin surgical institute in 1895–1903, only two cases of articular rheumatism were observed (Fröhner). That articular rheumatism does, however, occur in the horse has been proved beyond all manner of doubt by recent experiments (Pfeiffer, Tetzner, Altmann, Christiani, Fröhner, and others). Very few of the arthritic affections of sucklings belong to this category. It is, therefore, best to discuss them with pyæmic articular inflammation, with which they have often been confused.

Etiology.—The main source of articular rheumatism was formerly sought in colds, for the production of which the same factors were blamed (draughts, damp, cold, bad quarters) as in the muscular form. There is, however, no doubt that, as in man, so also in our domestic animals, articular rheumatism is an infective disease in which chill only acts the part of a predisposing cause. Proofs of its infectious nature are: the general feverish signs, as well as the initial stage characteristic of all infectious maladies, the simultaneous affection of several joints, often far removed from each other; the endocarditis which often also appears; its outbreak in model farms and byres, where taking cold is carefully guarded against; and the observation made by many authorities that articular rheumatism is indisputably connected with the retention and putrefaction of the after-birth. Auer noticed the disease almost exclusively in young cows after their second or third calf, and in two-thirds of all the cases observed (67 in all) could prove abortion or retention of the after-birth. He therefore regards the resorption of septic matter in the uterus as the direct cause of articular rheumatism. Dinter and Dessart also saw the disease exclusively in cows that had newly calved. We may therefore conclude that in cattle the puerperal uterus is the chief source of the infectious matter which produces this disease. Barthélemy observed an epizoötic articular rheumatism in goats. As in other

infective diseases, chill favours the penetration into the body of the infective matter and also its multiplication, by rendering it weaker and less able to resist, but the infective substance in question is not yet bacteriologically known. It is possible that various bacteria may produce the aspect of articular rheumatism, and that, therefore, the affection does not in this respect represent an etiological unity. At the present time, the streptococcus is generally regarded as the cause of articular rheumatism.

Puerperal Polyarthritis.—Articular rheumatism in cattle has been associated by many with the retention and decomposition of the placenta. Auer observed articular rheumatism only in young cows after their second or third calf, and in two-thirds of the cases (67) he was able to point to a previous history of either abortion or of retention of the after-birth. He assumes from this fact that articular rheumatism in these cases was due to the absorption of septic material by the uterus. Dinter and Dessart encountered the condition in cows only after calving. The inference drawn from these facts is, that the uterus is the principal source whence the poison, which gives rise to articular rheumatism in cattle, is derived. In Switzerland, however, this puerperal polyarthritis is regarded as a secondary, metastatic manifestation, quite distinct from articular rheumatism (Hess, Guillebeau, Ehrhard and others). According to Peauleau, Cadiot, Moussu, and others, the hip, knee and fetlock joints are more particularly affected in "post-partum arthritis"; and the condition is most frequently observed after an abortion, a difficult delivery, or a retention of the placenta. The inflammation is not, as a rule, suppurative; it is said to be due to the agency of the staphylococcus. The prognosis is unfavourable.

Bacteriology.—According to Wassermann, Meyer and others, the originating cause of articular rheumatism in man is a specific streptococcus, which may also give rise to multiple arthritis with high fever in animals. The source of infection was formerly believed to be a plasmodium which entered the system by way of the tonsils, articular rheumatism being frequently complicated with tonsilitis (according to Fiedler 158 times out of 655 cases). Edlefsen maintains that it is a miasmatic contagious disease which occurs most frequently when there is a decrease in the atmospheric pressure, and may be confined to certain localities ("rheumatic houses"). Secondary or predisposing causes are over-exertion and chill. Thiroloix and others state having proved the existence of a specific bacillus.

Post-Mortem Conditions.—The anatomical changes in the joints consist mainly in a serous synovitis and, in chronic cases, in inflammatory, degenerative modifications of the joints; a suppurative synovitis is but rarely observed. Generally, several joints are affected at the same time. When the course is acute, we find the synovial membrane highly

reddened, even permeated by hæmorrhagic areas, opaque and swollen, sometimes also thickened, and the synovial villi vascular and enlarged. The synovial fluid is increased, coloured red and often also opaque. The articular cartilages are at first reddened, later they turn yellow and display a rough, velvety surface. The tissues round about the joints also appear to be injected by hæmorrhages and serously infiltrated (periarthritis), the connective tissue especially is swollen and gelatinous, and the neighbouring muscles œdematous and softened. The articular ends of the bones are, moreover, hyperæmic and even hæmorrhagically infiltrated, and likewise their medulla. During the chronic course of the disease the synovial membrane becomes very much thickened, and the inner surface of the joint often contains a coating of connective tissue very rich in cells (arthritis pannosa); the articular cartilages undergo fatty disintegration, show an ulcerative loss of substance and are partially detached. In rare cases, an arthritis deformans is developed, as in man. Thus Moore noticed the bones of the root of the tarsus become anchylosed to the metatarsus in a dog suffering from chronic articular rheumatism, and also irregular formation of osteophytes. Among general physical changes endocarditis, pleuritis and peritonitis sometimes occur; other changes are of no moment.

Symptoms.—The most important of these is swelling of the joints, which often appears suddenly, frequently overnight, and may soon attain considerable dimensions. It generally attacks several joints on the same or on different legs, and seems to prefer the carpal, tarsal and knee-joints. The other joints are often affected in a distinctly ascending scale. A single joint is rarely attacked (mon-articular form). The swellings are hot, very painful, feel tightly stretched and in some spots fluctuating, and extend all around the joint. The adjoining parts (tendons, tendon-sheaths, muscles) are also implicated in the inflammatory process and are swollen. The animals consequently limp so much, that a non-professional observer might suspect a broken bone. The swellings may be so painful when touched for examination, or when the joints are bent, stretched or turned, that the animals can only with difficulty retain their feet. They therefore avoid every movement or even putting the affected leg to the ground, often lie down continuously, groan and grind their teeth, can only with difficulty be made to stand up, and sometimes not at all. Their

internal temperature is also at first raised and is on an average 1½° to 3½° Fahr. higher; the pulse is small and hard, and, in cattle, from 70 to 90 beats a minute. Both appetite and rumination are suppressed, the muzzle is dry, defecation delayed, and the secretion of milk much decreased, the milk itself being of a sour flavour and readily coagulating. We may also observe a striking loss of flesh in cattle, even after the first two days.

In addition to these customary symptoms we in many cases note peculiar complications. First, and especially in cattle, an acute endocarditis may develop, which sometimes leads to sudden death. Then appearances also of pleuritis, peritonitis, pericarditis and laryngitis may be observed, as well as inflammation of the tendon-sheaths. These complications, as also the exanthemata (urticaria) sometimes seen in man, cannot be understood except on the hypothesis of a general infectious disease.

The *course* is usually chronic, seldom acute. In solitary cases a retrogression of the inflammatory articular swellings sets in with abatement of the fever, and a decided recovery takes place in two to three weeks, though such a benign course of the malady is rare. Very often the cure is only apparent, and a relapse soon occurs. For instance, a severely affected joint may recover with surprising quickness in a few days, while another and distant one may be freshly attacked (migration of articular rheumatism). In by far the majority of cases the course is wearisome and unfavourable. The feverish signs disappear, it is true, but the disabilities and lameness remain in the joints. Chronic degenerative changes gradually develop in the latter, the animals go back in condition, yield less and less milk, suffer from digestive disorders and gastro-intestinal catarrh with very violent diarrhœa and grow by degrees weaker and thinner. The groups of muscles above the affected joints (those of the shoulder, etc.) are atrophied, the extremities become stiff and crooked, and the joints remain thickened. Matters may continue thus for two to three months or longer. Fröhner found that after treating a horse for seven weeks (1½ lbs. salicylic acid per week!), he was able to bring about a certain improvement, but no definite cure. Dammann noted a case of a horse which lasted eight months without cure. If the animals be not soon slaughtered, they often perish of exhaustion, their bodies being covered with sores brought on by prolonged lying.

As regards *differential diagnosis*, traumatic and pyæmic

arthritis must be taken into consideration, also meningitis, when the vertebral articulations are affected.

Therapeutics.—So long as the nature of the infective matter is not more fully known, we can only recommend by way of prophylaxis the avoidance of the most important contributory cause of articular rheumatism, viz., chills. Horses especially must be rubbed down, clothed or walked about after violent exercise until dry. In cattle an early removal of the after-birth and thorough disinfection of the uterus must in all cases be remembered. Internally salicylic acid and salicylate of soda are specific and admirable remedies. For doses and method of administration, see the Treatment of Muscular Rheumatism (daily dose of salicylate of soda for cattle and horses 3 to 4½ oz.; for dogs and pigs ½ to 2 drams). Instead of salicylic acid we may give salol, salipyrin, salophen, antipyrin, phenacetin, antifebrin and similar new remedies, of which by far the cheapest is antifebrin. To promote absorption of the arthritic exudations and elimination of the infectious matter through the mucous membranes of the intestines, laxatives may also be administered, especially tartar emetic and large doses of neutral salts. Iodide of potassium may also be tried as a re-absorbent. Among *external means* we suggest warm wrappings about the affected joint, after first rubbing it with an ointment of grey mercury, or of carbolic acid, iodoform, chloroform or camphor. In chronic cases with delayed resorption, we may have recourse to sharp inunctions of the joint (tincture of iodine, cantharides ointment, iodide of mercury). Food must be carefully regulated, movement of the animal avoided and its stable kept warm and sufficiently ventilated.

PYÆMIC AND SEPTIC ARTHRITIS ("LIMPING") OF SUCKLINGS (JOINT-ILL).

General Remarks on the Term "Limping" (*Lähme*).—Under the name of "Limping" a series of ailments in sucking animals (foals, calves, lambs, sucking pigs and puppies) has long been included, which, although essentially differing in cause and nature, are yet alike in manifesting themselves by serious disturbances of locomotion. The term limping is therefore only a generic name, which should no longer be retained,

but replaced, if possible, by more exact scientific nomenclature, the necessity for which has long been recognised by veterinary authorities, and as early as 1839 the "limping" of foals was referred to by Träger as including an entire dictionary of diseases. Thanks to Bollinger, we can now better differentiate the various diseases so designated, and would therefore indicate the following:

1. *Pyæmic or septic articular inflammation* which develops by metastasis from a septic infection of the navel-wound. This septic-pyæmic polyarthritis is, according to Bollinger, the most serious and frequent cause of the limping of sucklings.

2. *Fatty degeneration of the body muscles*, especially in sucking pigs.

3. *Articular rheumatism and muscular rheumatism.*

4. *Rickets*, particularly in sucking pigs.

5. *Tetanus and cerebro-spinal meningitis*, especially among lambs.

6. *Tabes*, a disease of foals, which is probably identical with tuberculosis of the intestinal and mesenteric glands.

Various other diseases to which sucklings are liable, *e.g.*, intestinal catarrh, broncho-pneumonia, pleuro-pneumonia, general debility, weakness in the extensor muscles of the extremities with consecutive contraction of the flexors (in foals and dogs), etc., are also popularly known as "limping."

Etiology of Pyæmic Arthritis.—The causes of this form of inflammation, which occurs especially among foals and calves and also among lambs, must be sought in a purulent, ichorous inflammation of the navel and its surroundings, with thrombosis of the umbilical vein, suppurative disintegration of the thrombus and distribution of its waste matter by the blood. The starting-point is an infection of the navel-wound by dirt from the floor of the stable. Uffreduzzi cultivated two separate bacteria—a micrococcus and a bacillus—from the bodies of calves which had died of this disease. His successful inoculation with this bacteria produced not only pyæmic, but also septicæmic symptoms, so that it may be presumed that from the infected navel-wound both pyæmia and septicæmia may develop. Buch found in the liver micrococci which had a likeness to the diplococci of pleuro-pneumonia. Turner discovered the same bacteria in the membranes of the ovum of brood-mares suffering from epizoötic abortion, as in the organs of foals that had died of "limping." Cultures of it injected

into the vagina of pregnant mares produced abortion, with arthritis in the aborted foals.

The cause is to be found in connection with the very insufficient care which is bestowed upon the navels of our domestic animals. Bollinger affirms that a thick, gelatinous character of the umbilical cord predisposes to this affection; the same, too, may be said of wrenching the umbilical vessels, or tearing them off too short, contusion of the wound, or injury to the abdominal walls. According to Gmelin, the predisposition must be sought in the navel itself. He states that necrotic processes take place quite normally within the umbilical ring; that the umbilical arteries become plugged by thrombi, the central ends of which are connected with the circulating blood and the peripheral ends with the outer air and its neighbouring necro-biotic processes, so that the peritoneum which covers the inner navel-ring is drawn into direct sympathetic affection with all the morbid processes going on within the umbilicus. Births in early winter time, necessitating a long sojourn in the stall, greatly increase the risk of infection from the manure-soaked ground.

The disease sometimes appears enzoötically in herds and breeding stables. According to Hering, of 187 sucking foals which died during 15 years in the provincial breeding stud at Marbach in Württemberg, 85 perished of "limping." Gmelin gives some interesting details concerning the history of the disease in this particular stud. Among the stud documents, which date from 1767, is a report to the Duke Karl Eugen of Württemberg, in which particulars are given of the occurrence of the disease. The stud suffered severe losses in 1808 and in 1852–63; between the last-named dates 119 foals died. From 1871–1880, 35 foals died, an annual loss of 5 per cent. From 1881–1889, 18 foals died, an average loss of 4 per cent. annually.

The question of *hereditary transmission* of "limping" was in former times universally answered in the affirmative. The contrary opinion was first energetically proclaimed by Hering in 1863. Even recently, professional men have maintained that an intra-uterine infection of the fœtus by the mother animal may occur, in addition to any subsequent affection of the navel, because the navel of foals suffering from "limping" very often shows no morbid signs. This argument does not seem to us to be valid, for, on the one hand, the navel may remain apparently sound in spite of infection in the body, an analogy to which fact may be found in the womb during paresis of parturition, and, on the other hand, the septic navel-affection may heal, while the pyæmic processes go forward in the body. How else can we explain the undoubtedly favourable results of prophylactic care of the navel (Bollinger, Gmelin)

if there be an intra-uterine, hæmatogenous infection? The possibility of such intra-uterine infection does not seem to us to be proved, yet it may easily happen that some foals inherit a certain predisposition to pyæmic polyarthritis, in so far as they are burdened with a low vitality and feeble constitution, which may be transmitted from their mothers. This congenital weakness was also included in the term "limping."

In adopting a standpoint opposed to the hereditary transmission of "limping," we base our views upon investigations recently undertaken by Ostertag. His conclusions are opposed to those of Sohnle, who assumes an intro-uterine infection of the fœtus by the mother and attributes the same cause, namely, a specific staphylococcus, both in enzoötic abortion and in the "limping" of foals. Ostertag, on the contrary, is of opinion that the "limping" of foals is not due to a specific cause, but that it is a form of ordinary sepsis and pyæmia; and may be brought about by the agency, not only of the staphylococcus, but also of the streptococcus. He holds that there is no ground for the assumption that enzoötic abortion and the "limping" of foals originate from a common etiological agent, and points to the fact that congenital "limping" or polyarthritis has never been observed. The rapid onset of the condition in new-born foals cannot be taken as a proof of intra-uterine infection; on the contrary, Sohnle's own experiments show that a metastatic articular rheumatism, which is also the earliest symptoms of "limping," may develop in the course of the first day after birth.

Bacteriology.—Uffreduzzi has succeeded in obtaining two different schizomycetes from the bodies of calves which had died under symptoms of pyæmia. They are a micrococcus and a bacillus, and he has been able to re-inoculate with both. In addition to pyæmia, he also observed appearances of septicæmia; hence it is to be inferred that the infected umbilical wound may give rise to both conditions. Buch found micrococci in the liver, which resembled the diplococcus of contagious pleuro-pneumonia. Dobes reports similar findings. Turner found bacteria in the membranes of brood-mares subject to enzoötic abortion, which were identical with those discovered in the organs of foals which had died of "limping." Cultures of these, injected into the vagina of pregnant mares, produced abortion, and, in the aborted foals, arthritis. The experiments of Gasperini and Fogliata were attended by similar results.

Post-Mortem Conditions.—The phenomena revealed are essentially those of suppurative polyarthritis, with other changes characteristic of the pyæmic process (pyæmic form); in other cases the aspect of septicæmia is more pronounced (septic form). The navel-wound, if not already healed, shows signs of inflammation and suppuration, the umbilical cord and surrounding region are swollen, the edges of the navel-ring are ulcerated, within the inner umbilical ring abscess-formation is found, and through the opening of the navel drops of pus can be squeezed. With this omphalitis—which, as

stated, may have healed and left no external morbid signs—we find, on going deeper, a suppurative thrombo-phlebitis and thrombo-arteritis umbilicalis, with thrombosis of the portal vein and its branches in the liver. In the joints the synovial membrane is injected, swollen and thickened, the synovial fluid is opaque, increased in quantity, mixed with flaky coagulæ and later of purely purulent character (empyema of the joint). The articular cartilage is ulcerated or destroyed, sometimes even the bony tissue is necrotic and the articular capsules perforated. Abscesses form round about the joints between the tendons and muscles, the latter being purulently infiltrated. The muscles over the affected joints have undergone fatty degeneration. The most marked lesions are found in the knee and hock joints and the joints attaching the head to the neck. Dissection also reveals metastatic foci in almost all organs, especially in the liver, lungs, brain, kidneys, muscles and subcutaneous cellular tissue. In the liver these foci range in size from a millet-seed to a cherry or larger ; and in the lungs from a millet seed to a hen's egg. They are at first dark red and later turn yellow (so-called suppuration of the lungs). Pleuritis, endocarditis and pericarditis are also observed, as well as broncho-pneumonia, inflammation of the tendon-sheaths, peritonitis, cystitis, suppurative iritis, ophthalmia, meningitis, etc. Finally, the heart, liver, kidneys and muscles are in a state of fatty degeneration.

Symptoms.—Pyæmic arthritis in the greater number of cases develops in the first days or weeks after birth. Of 67 foals, says Hering, 40 (or 70 per cent.) sickened and died in the first three weeks of life. Bollinger, taking his figures from another breeding establishment, says 75 per cent. Usually a few general symptoms precede the appearance of the arthritic affection, such as fever, diminished or suppressed desire to suck, etc. ; but these may be absent. The swellings on the joints generally develop very quickly. The knee and hock joints, as well as the elbow, hip and shoulder are most frequently attacked ; also the fetlock and coronet, while even the joints of the spine and ribs do not escape. The swelling is hot and painful, very tense, and at first often disappears rather quickly. In its later course it has a tendency to form abscesses, the pus within the joint penetrating outwards. Generally several or even all the joints of the body are simultaneously inflamed. The animals grow very lame, are afraid to touch the ground

with the affected legs and lie down a great deal. To these symptoms diarrhœa is soon added, the fæces being greyish-white and bad-smelling, and with the diarrhœa constipation alternates. The animals grow very thin, weak and debilitated. According to the position of the metastasis, various complications may be observed (inflammation of the lungs, sudden swellings in different parts of the body, cerebral phenomena, blindness, etc.).

The *course* in foals is generally acute; the average duration of the illness being from two to three weeks, but a fatal end may occur even in a few days. According to Gmelin's observations, the foals which died of pyæmia attained an average age of 26 days; the disease declaring itself on their 17th day and killing them after an average duration of nine days. The *prognosis* is very unfavourable, estimates of mortality ranging from over 50 to 90 per cent. of those afflicted. The remainder recovered very slowly, continued for a long time to be sickly and were often backward in growth. The swellings on the joints and the stiffness which they cause are also long in passing away, or may even become permanent.

In *calves* the prognosis appears more favourable, the mortality, according to Strebel, being only 35 per cent. The course with them is, however, slower, sometimes even chronic, and the pyæmic affection is less widely diffused through the body. Sometimes one joint only, that of the patella, is affected; and this was noted, according to Strebel, in older animals (of three to five months). Probably this more favourable course in calves is due to the circumstance that, as compared with foals, their umbilical arteries tear within the abdominal cavity and can therefore retract further, whereby their thrombi are better protected from infection (Gmelin).

Differential Diagnosis. — In order to distinguish pyæmic arthritis from acute articular rheumatism—two complaints very easily confounded—the following points may be of importance. First of all, the development of a disease in the joints of sucklings a few days or weeks old proclaims pyæmic arthritis, especially if a suppurative inflammation of the navel can at the same time be detected. Moreover, the formation of abscesses in articular rheumatism is so rare an occurrence, that it may always be taken as a sign of the disease under consideration. Finally, articular rheumatism seems to be much less common among sucklings.

Therapeutics.—In accordance with the pathogenesis of the ailment, its treatment is especially prophylactic. Bollinger recommends above all things strict attention to the navel, careful ligature of the umbilical cord, antiseptic treatment of the navel-wound, with a protective bandage round the belly; or, if necessary, the bandage may be replaced by daubing with tar. Gmelin advises, after ligaturing the umbilical cord, painting the cut surface with concentrated carbolic acid until (four to five days) the rest of the cord assumes a parchment-like appearance. Then clip off the navel-stump with scissors and paint the wound daily (seven to eight days) with sublimate gelatine (1 per cent.) until it heals. After introducing this plan the disease completely vanished from a stud where it was formerly very prevalent. The stalls must also be kept very clean, dung and urine removed, the floor disinfected and good, dry straw provided. In the disease itself antiseptic treatment of the umbilical region must be adopted (cleansing with creolin or carbolic solutions, application of a 10 per cent. ointment of creolin, carbolic or iodoform). Also anti-febrile and anti-zymotic remedies must be used, especially camphor (subcutaneous injection of spirits of camphor in doses of 20 to 80 min.); also stimulants (alcohol, wine). The treatment of the affected joint is purely surgical. Above all, the pus must be removed as soon as possible by puncture or incision.

Fatty Degeneration of the Muscles in Sucklings.—An affection of the motor apparatus is described under this name by Fürstenberg, Roloff, and Repiquet. It occurs with the greatest frequency among English swine and well-bred lambs, and, more rarely, among foals and calves. It is, however, only one of the symptoms of a general congenital fatty degeneration, and its description should properly be included under the heading *Obesity*. It is developed during the second half of the fœtal life, and its causes may be sought in an inherited predisposition and in a tendency to fatness in the mother, found especially among breeds which reach maturity early; also in the mother's resting too much in the stall, combined with over-richness of feeding. Fürstenberg also condemns breeding from animals too closely related. The *anatomical changes* consist in general anæmia, fatty degeneration of the body muscles (which often cannot be recognised as such, but shine like lard or look as though cooked), also in fatty degeneration of the liver, kidneys, brain, pancreas and of the glands of the intestinal canal. The *symptoms* reveal themselves in general weakness, dull, sluggish movements and even inability either to walk or stand. In severer stages of the disease the animals lie in complete apathy and often die speedily without any further complications or after attacks of diarrhœa, spasms, and paralysis. The ultimate cause of death is frequently œdema of the lungs. *Treatment* is only prophylactic. An introduction of fresh

blood by frequently crossing the breed, exercise and more restricted feeding of the mothers during pregnancy, are the most important matters. Any medicinal treatment of the young animals is fruitless. Castellant describes a case of fatty degeneration of the entire muscular apparatus of the rump in an adult ox, which was of so extreme a nature that, of 440 lbs. of meat, 130 lbs. were converted into tallow.

Infectious Articular Inflammation in Young Geese.—Lucet has observed an articular inflammation in goslings (caused by staphylococcus pyogenes aureus) which appears sometimes sporadically and sometimes enzoötically. It manifests itself in lameness, diarrhœa, fever, much lying down, swelling of the joints and in death after from one to three days. More rarely the course is sub-acute and chronic (formation of anchyloses). The disease can be induced in goslings by inoculation with staphylococcus pyogenes aureus.

OSTEOMALACIA AND RICKETS.

General Notes on Diseases of the Bones.—Those diseases of the bones which belong to the realm of internal pathology, of which osteomalacia and rickets are by far the most important, might very well be relegated to the chapter on general disturbance of nutrition. On the other hand, it has long been the custom to rank them among diseases of the locomotory apparatus because, during life, their most prominent symptom has been a defective power of motion. A purely etiological classification is still impossible, as, in spite of numerous experimental investigations, their etiology is not yet sufficiently cleared up. Neither has pathological anatomy hitherto been able to furnish a clear survey of the pathogenesis of these bone diseases. It may perhaps be best to classify those of internal pathology into two great groups:—

1. The *bone diseases of the earlier period of life*, of which *rickets* is the principal manifestation.
2. The *bone diseases of later life*, among which *osteomalacia* s the most important.

As to the relation between rickets and osteomalacia opinions by no means agree, though the two diseases are obviously related, as is shown by the fact that at certain times they appear simultaneously in the same stable or the same herd and that both may be experimentally and simultaneously produced in the same animal; the prime cause of each is to be sought in a deficient supply of lime. The pathological changes in the bones show great differences, but if we bear in mind that, even normally, there are considerable structural differences

between a growing and a fully-developed bone, we may assume that the varied pathological aspect of rickets and osteomalacia may arise solely from the different ages of the bones affected. According to this, rickets may be regarded as osteomalacia modified by the condition of growth in younger bones. We therefore define the relation of both as follows :—

1. *Osteomalacia is a re-softening of the bones of mature animals* in consequence of resorbtion of their lime-salts (halisteresis).

2. *Rickets*, on the contrary, *arises from the bones of young animals remaining soft* owing to deficient calcification.

Experimental Investigations into the Etiology of Rickets and Osteomalacia.—Many experiments have been made to ascertain if possible the causes of these two conditions, but the results cannot be considered satisfactory. Nevertheless, as they have given considerable help towards an understanding of the diseases, we must devote a few sentences to their discussion. The following three theories, which are briefly known as the "inanition," the "inflammation" and the "acid" theories, have become specially important for pathogenesis.

1. *The theory of inanition* assumes as the chief cause of both diseases of the bones a deficiency of lime in the food. Roloff produced decided phenomena of rickets in young dogs and pigs by continuously feeding them on food that was poor in lime, and those of osteomalacia in a full-grown goat and sheep. The rachitic symptoms were: lameness; curvature, crookedness and thickening of the bones; swelling of the costal cartilages (rachitic rosary); contraction of the pelvis; thickening of the joints; so-called double-joints; bear-footed, bandy-legged, or cow-hocked attitude; turning of the knee-joints outwards; bending of the hocks; curvature of the spine; infraction; halting or staggering gait; debilitated condition; painful sensibility to touch; symptoms of pica, etc. E. Voit induced symptoms similar to these in puppies by means of a long-continued diet deficient in lime. There was the same backwardness in the development of the skeleton, as well as a lack of firmness and thickness about the individual bones, together with all the characteristic symptoms of rickets. These were: epiphyseal swelling; inflection of the ribs and of the shoulder-blades; curvature of the extremities; clumsy, painful gait, in every degree of intensity up to inability even to move the limbs.

The results of the experiments undertaken by Roloff and

Voit are confirmed by the clinical experience of practising surgeons. A poverty of lime in the food is regarded as the principal cause of both rickets and osteomalacia. Osteomalacia in pregnant and milch-cows, moreover, appears to stand in etiological relationship to the increased withdrawal of lime from the body. Finally, both pathological conditions may be overcome by giving an increased quantity of lime.

The results of these experiments, which are confirmed by those of Chossat, Milne-Edwards, Lehmann, Miwa and Stölzner, are, however, not borne out by those of Weiske. The animals which he treated by means of a diet deficient in lime certainly died, but not from rickets. It has been urged that Weiske's animals perished, not from the results of an insufficiency of lime, but from inanition, before the bones had time to become pathologically changed. An insufficiency of lime does, however, constitute an important factor in the etiology of both diseases.

2. *The inflammation theory* assumes that both these diseases of the bony tissue are due to a substance floating in the blood, which sets up inflammation therein. The starting point of this theory was a series of experiments by Wegner, who, after prolonged administration of very small quantities of phosphorus, observed a thickening of the bones on the diaphyses. From this fact Kassomitz concluded that in rickets also a substance must be present in the blood resembling phosphorus in action, which causes inflammation of the bones with increase of vascularity, during which, and as a result of dilatation of the blood-vessels, the deposit of lime is hindered. Also, in consequence of the decomposing action of the carbonic acid of the blood upon the bones, their tissue is broken down. Kassowitz succeeded by giving phosphorus in larger quantities, in obtaining bone-modifications precisely resembling the conditions of rickets. Respecting the nature of this inflammatory substance nothing further is known ; but we must assume it to be an infective substance which acts from the blood (hæmatogenous inflammation of the bones). Virchow has already declared osteomalacia of man to be a parenchymatous inflammation of the bones.

According to Winckel, we have to deal with an ostitis chronica with de-calcification of the bones from within, and simultaneous, localised new building up of bony tissue. Stölzner affirms that the defective calcification in rickets does not result from an insufficiency of lime in the food, but from a morbid condition of the bony tissue itself.

Petrone's suggestion is that osteomalacia represents an infectious disease, produced by the micro-organism of nitrification discovered by Winogradsky, and that this bacterium generates nitrous acid in the bones, which dissolves out their lime-salts. Delcourt believes rickets to be due to the action of potash salts (potatoes).

3. *The acid theory* is based on the assumption that during catarrh of the stomach and bowels, which is peculiarly frequent in young animals, an abnormal quantity of lactic acid is generated from the milk and carbohydrates absorbed, and that this acid, after having passed into the blood, dissolves out the lime-salts of the bones and causes proliferation of the bony tissue. Heitzmann tried to prove this theory by producing rickets in animals by persistent subcutaneous injection of lactic acid and feeding them on the same substance. Roloff, Heiss, Tripier and others failed to confirm his statements by the experiments they also made with lactic acid. Roloff holds that, as Heitzmann avoided at the same time all foods containing lime, he obtained his results not by the supply of lactic acid, but by withholding lime. The proofs, which other authorities are said to have obtained, of the presence of lactic acid in the bones and urine in rickets, have been shown, upon closer investigation, to be inconclusive. Virchow, Hofmann and others also discovered that fresh rickety bones show an alkaline and not an acid reaction, and a careful analysis of the urine also failed to trace any lactic acid therein. Finally, the lactic acid, on passing into the blood, must probably be quickly decomposed into carbonic acid and water, or into lactic salts. Other strongly acid foods, such as distillers' or brewers' refuse, however large the quantities in which they may be consumed, do not induce rickets. The experiments of Siedamgrotsky and Hofmeister supply the right material upon which to base an estimate of the lactic acid theory. These authors were also unable to produce typical rickets or osteomalacia by the administration of lactic acid, but found that the amount of mineral substances in the bones was somewhat reduced. They infer from this that the solvent action of the lactic acid cannot be very marked; and that, although the importance of lactic acid as a factor in the causation of diseases of the bones is considerably reduced by the results of their experiments, it is by no means to be regarded as a negligible quantity. From these and other observations it is plain that lactic acid plays only a very secondary part in the etiology of bone diseases.

A.—OSTEOMALACIA OR BRITTLENESS OF BONE.

(*Softening of the Marrow, Osteopsathyrosis.*)

Occurrence and Etiology.—Osteomalacia generally described in veterinary literature as "softening of bone" occurs chiefly in cattle, less frequently in sheep, goats and pigs. It is rare in horses. It has also been noticed in zoological gardens

among giraffes and a few kinds of birds, is one of the oldest known of diseases among cattle, and was accurately described by Vegetius. It seems to be located in certain districts, where in some seasons it becomes almost epizoötic in its spread, and attracts the attention of Government. It not unfrequently appears sporadically in single farms or stables. Milch-cows are most freely attacked ; working oxen or young kine much more rarely, also old cows. The periods of pregnancy and lactation greatly hasten the development of the disease (osteomalacia puerperalis). The more milk is yielded, the more intense is the ailment ; while if the milk-secretion be suppressed, at once an improvement is generally seen. In many districts the cows regularly fall ill 6–8 weeks after calving. These facts point to an increased output of lime on the part of the mother as being a cause of the disease.

Besides this, a deficiency of lime in the soil and fodder are known to be contributory factors. Moor and turf lands, sandy soil poor in chalk, plutonic rocks and soil deficient in phosphoric acid on the one hand and sour grasses and pastures on the other seem to be the most frequent sources of osteomalacia. To these we must add very dry summers, in which there is a lack of fluid to dissolve the lime-salts in the ground and convey them to the grass roots ; also feeding on potatoes and turnips, which are vegetables deficient in lime. The chemical investigations of Cantiget are interesting on this point. Tests of various soils gave a lime or chalky average in districts free from this complaint of 245 ; of only 10–25 in those occasionally affected ; and of 9 in those frequently visited by the disease. The corresponding figures for phosphoric acid were 4.1 and 0.9. In correspondence with these figures, we often find osteomalacia on those farms where very bulky food is served, such as straw and all kinds of waste of low nutritive quality. Moreover, the small degree of solubility and absorbability possessed by lime-salts, even though, according to analysis, they may be relatively abundant (as, *e.g.*, in fodder of very woody fibre), might also be a predisposing cause of osteomalacia. Finally, the water may assist its development by being insufficiently calcareous.

Dirty, ill-kept stalls and chill (osteomalacia rheumatica) were formerly regarded as the principal causes of osteomalacia. Whether these conditions actually give rise to osteomalacia is the more doubtful, when we consider that the disease may occur under faultless hygienic conditions. Nevertheless, where unsatisfactory nutritional methods prevail, chill and bad stabling

may easily hasten the onset of the disease. It has recently been pointed out that osteomalacia occurs less frequently among pasturing animals than among those kept continuously in the byre. Animals at grass are able to satisfy their "earth-hunger" directly by the ingestion of sand, earth, etc. (Höhne).

Animals recovering from some other disease (foot-and-mouth) may be more readily infected. On the action of infective matter nothing is at present known, but many etiological factors may possibly co-operate here, as also in rickets.

In man we distinguish a juvenile, a senile and a nervous form of osteomalacia. According to Zschokke, osteomalacia occurs sometimes in puppies which is identical with the form in children.

Post-Mortem Conditions.—The pathological changes found in the bones consist mainly in a decalcification and softening from within outwards of the normal bone, with hyperæmia, conversion of the bony substance into a fibrous, soft mass, fatty degeneration and atrophy of the bone cells, enlargement of the medullary cavities, an increasing thinness of the cortex and a change of the medulla itself into a soft and gelatinous mass. The most marked alterations are found in the bones of the trunk and of the upper extremities. The aspect varies according to the stage of the disease.

1. *At the beginning* and in slighter cases we discover nothing morbid on superficial examination (Roloff), but closer investigation reveals an increased amount of blood in the affected bones and enlargement of the vessels and of the canals which contain them. A section of the bones shows small dots of blood, the marrow permeated by numerous little extravasations, and the bony tissue round the dilated Haversian canals exhibits slight changes.

2. *In later stages* the hyperæmia increases; the outer surface of the cortex as well as the bone-section are dotted with blood, the marrow becomes hyperæmic and penetrated by hæmorrhages. The medullary cavity itself is enlarged. Upon the diploë and inner surface of the cortex are numerous fine bony plates detached from the mass. The bone also is easier to saw or cut, and its ring has lost its clearness. The microscope shows that the homogeneous structure of the bony substance has vanished; that in some spots it appears clearer, the bone cells are enlarged and transparent, have assumed an oval, round or even polyhedral shape, their prolongations disappear and they

gradually become transformed into fat cells. The whole represents a transformation in parts of the bony substance into osteoides and finally into medullary tissue.

3. *In its most severe forms* the hyperæmia of bone and marrow continues to increase; the substance of the cortex becomes spongy, opaque and soft; cortex and diploë tend more and more to vanish, while the medullary substance grows, presses the cortex from within outwards and penetrates also into the epiphyses. Fractures of the bones then occur. The marrow itself, if general disturbances of nutrition set in at the same time, becomes soft, gelatinous, even watery and has a dirty-yellow colour. The bones diminish in specific gravity, their lime contents being by this time lessened by more than half, but their watery contents increased. In addition to these processes in the bones, we sometimes find an accumulation of reddish fluid about the joints. Other organs are at first not pathologically changed. Later on we find, with increasing emaciation and weakness, the signs of cachexia; a hydræmic condition of the blood, transudation in the bodily cavities, general poverty of fat, flabby and discoloured muscles and gelatinous infiltration in the cellular tissue.

Symptoms.—Osteomalacia does not, as a rule, reveal itself from the beginning in external signs. No outward indication of its presence may appear for a long time in spite of serious changes in the bones. Thus there may be no pain or emaciation, and the milk-yield may be sustained for a lengthened period. Slight disturbances of digestion often precede the outbreak of the disease, and we notice also a frequent desire to lick various objects; this sign may, however, be absent. In cows advanced in pregnancy the disease may appear suddenly. Usually it is only the advanced stage which is brought to our notice. We observe then a painful, stiff gait, often on all four feet together, and this lameness sometimes appears suddenly after exertion or a lengthy march. The animals shift from foot to foot, sway to and fro, twitch with their hind legs, evince pain in getting up or lying down, in evacuation or urination, or when pressed upon the shoulders, withers or loins, and at last remain permanently lying. Goats especially seem to be completely lamed and show extraordinary pain on the slightest touch. Under some circumstances these signs are complicated with swellings on the joints with cracking sounds therein, and all the symptoms of articular inflammation, which are supposed,

however, not to develop in animals at rest, but only in those which have had exercise, being caused by strain upon the tendons and ligaments, with loosening of the periosteal adhesions and consequent spread of inflammation to the synovial capsule. Later on, fractures, fissures and dislocations occur on the slightest provocation, as in getting up, lying down, turning, slipping and during parturition. Fracture of the pelvis or ribs is most common, the former occurring especially on the ileum near the hip-joint (acetabulum). Several fractures are frequently found in the pelvis, and in one cow Maris saw no fewer than fifteen. All these are more or less painful and show little tendency to unite. The much-disputed possibility of the formation of callus seems to have been proved by Roloff's observations.

The ailment generally lasts several months, and during its later course emaciation sets in; the skin grows dry and hard, appetite falls off, feebleness increases and skin ulceration is brought on by continued decubitus. Hering also observed in several cases impetiginous eczema of the skin. The animals finally perish of extreme cachexia.

Osteomalacia of Horses.—This is much rarer than in cattle owing to the animals being better fed and receiving more grain-foods, which are richer in lime and phosphorus. Continuous poor feeding (chaff, bran, bad hay, straw) and deprivation of oats, combined with damp, chilly stables, are the chief causes of the complaint in horses. The diseases in these animals which are usually described under the names of "bran-sickness," "bean-sickness" (pollard sickness), and "osteoporosis," should be classed as osteomalacia. Probably rickety foals also sicken later of osteomalacia. The *symptoms* reveal themselves partly in a liability to fracture of bones (particularly of the spine), and partly in swellings of the upper and lower jaws with loosening of the teeth; also in stiff, straddling attitudes, much pain when moving; periodical lameness; weakness in the loins; sinking of the back; riding-stiffness (saddle-stress, resulting from osteitis and ankylosis of the spinal column); arching of the breast so as to look quite tub-shaped; thickening of the osinnominatum; thickening of the joints, especially of the knee, hock and coronet joints (osteomalacic knee-cap); and sensitiveness of the muscles to touch. *Post-mortem* shows the same changes as in cattle. In a case described by Baltz the entire vertebral column and the ribs were so soft that they could easily be cut with a knife. The transverse processes of the vertebræ could be rolled up like strips of pasteboard. Stiegler observed symptoms of rheumatism in a seven-year-old East Prussian horse. They were lameness; thickening of the hip, knee and fetlock joints; pronounced swelling of the left hind coronet joint (knee-cap); sinking of the back-bone and extreme lassitude. The autopsy showed that all the bones had undergone softening and were changed into a spongy mass,

the ends of the bones at the joints being partially destroyed. Elliott observed enzoötic osteomalacia (osteoporosis) in the island of Hawaii which annually carried off one hundred horses and mules. He considers it to be a climatic disease, induced mainly by damp. It is also said to occur in connection with wounds. The symptoms were lameness, emaciation, and swelling of the bones of the upper and lower jaws; the autopsy showed softening of all the bones.—*Treatment* is the same as for cattle.

Prognosis.—The forecast in osteomalacia greatly depends on whether a change in the animal's outward conditions be possible or not. Very advanced cases may be cured if a change of fodder, a transfer to another district or a rational medicinal treatment (calcium-phosphates, phosphorus) be possible. If circumstances do not permit of such a change, which, for financial reasons, is often the case, then, unless the animals are early slaughtered, they must be regarded as lost, especially if fractures have already occurred. Occasionally an improvement sets in after the period of suckling, but lasts only until the breeding time comes round again. According to Krabbe, out of 3,240 cows suffering from osteomalacia in 1877 in Norway, 281 were slaughtered as incurable (9 per cent.). Bougartz gives the proportion of recoveries at only 20 to 25 per cent.

Differential Diagnosis.—The recognition of osteomalacia in its fully developed stages, when fractures have already occurred, is not difficult. In the earlier stages, when the signs are less characteristic, it may be confused with muscular and articular rheumatism, the more so as complications with these two diseases are not rare. When osteomalacia appears sporadically, a positive differentiation between them is at first often impossible; but when its spread is epizoötic, a recognition is greatly facilitated by consideration of the etiological factor: According to Anacker, bones affected by osteomalacia give out on percussion a clear and hollow tone, in contrast to the deep and full-ringing note of sound bones. In goats actinomycotic swellings of the head-bones were formerly taken for osteomalacia; but the very situation of the disease and the varied nature of the lesions disprove this supposition.

Therapeutics.—The treatment of osteomalacia demands, first of all, the removal of its causes. Above all, a change of food must be made and its quality improved. Especially to be recommended are the foods richest in lime, such as leguminous foods, oats, clover-hay, oil-cake, and the straw of beans and

peas. A change in the drinking water may also be needful. The best plan is to transfer the animal to another district richer in suitable fodder, but when that is impossible, there is no alternative but to gradually improve the soil and its crops by rational manuring and draining. The best manures for this purpose are bone-meal and superphosphate of lime. Where the animals have been kept in confinement, they should be turned out to grass.

Internally, phosphate of lime must be administered in its most digestible forms. The best is the prepared (macerated) bone-meal, which may be mixed with the food in daily doses of ¾ to 1½ oz. (a tablespoonful with each feed). If this cannot be procured, give the same doses of calcium phosphate. Phosphorus itself may be given in small and long-continued doses (daily ⅛ to ¾ grain per cow, in oily solution). Occasionally, however, both phosphorus and calcium phosphate are without result. In such cases the employment of digestive stimulants will promote the assimilation, not only of food, but also of the lime. With these the absorption of lime may be assisted by stimulating appetite. Harms speaks highly of the action of spirits of salt (hydrochloric acid). Roloff suggests only partly milking the cows to prevent their losing too much lime; but a much better plan would be to withdraw them altogether from breeding while the disease lasts; economical reasons, however, often render both suggestions impracticable.

Since 1888 ovariotomy has been adopted for osteomalacia in women, partly with good results. This plan has, however, been but little tried among cows up to the present; but as ovariotomy is well borne by cattle, we would suggest that, where the disease makes its appearance epizoötically, this method be experimentally tested.

Osteoporosis.—In pathological anatomy this name is given to an enlargement of the Haversian canals, accompanied by atrophy and resorption of the compact bony substance, whereby the bones are rendered porous (rarefying inflammation of the bone). This rarefying osteitis occurs in several diseases. We find it in the course of osteomalacia and of actinomycosis. As Marcone has recently pointed out, osteoporosis, which occurs with particular frequency among horses, is really osteomalacia. For this reason we do not think there is any justification for regarding osteoporosis as a distinct clinical form of disease. Resorption of the bone by the medulla is also called eccentric atrophy of the bone; whereas with external resorption we have set up a concentric atrophy of the bone. Both are found with osteomalacia; but they are no more independent diseases than their related form, osteoporosis.

B.—RICKETS.

Soft Bones: Dwarfed Growth. Weak Limbs.

Occurrence and Etiology.—Rickets is a disease exclusively of youth, and is most frequently met with in young pigs and dogs, rarer in foals and calves ; it has also been observed in lion cubs and poultry. Among 70,000 dogs treated at the Berlin Veterinary Institute in 1886–94, there were 236 cases of rickets (0.3 per cent.). In some of these the condition is congenital (fœtal rickets). Thus B. Marchi describes congenital rickets in cattle in the Val Chiana (Tuscany). The causes correspond generally with those of osteomalacia, and lie chiefly in a lack of lime in the food, a fact abundantly proved by experiment, with food containing an insufficiency of it. First comes milk deficient in lime, such as is supplied by mothers suffering from osteomalacia. Among pigs it arises frequently from feeding with large quantities of innutritious and indigestible food poor in lime, such as kitchen refuse, or from an exclusive diet of potatoes, as a consequence of which rickets is always most common among young pigs after an abundant potato harvest. In dogs rickets is often caused by feeding them on bread, potatoes and dog-biscuits alone. An exclusively meat diet may have the same result, if no bones be given also, as was noticed by Röll with young lions. Feeding with bran alone is a most frequent source of rickets in foals (so-called bran-sickness), and may be experimentally produced. A lack of lime constituents in the soil is also an indirect cause of rickets. Thus Utz noticed that the disease was much rarer upon shell-limestone than upon red-sandstone, granite and gneiss.

The effect of a diet deficient in lime is much increased and accelerated by bad bringing-up. The endeavour by all means to fatten pigs before their skeleton has well formed, their unnatural forcing, the overcrowding of their sties, the foul air, lack of sunshine and absence of all natural exercise, amply explain why rickets is so much commoner in sty-bred pigs than in those which feed at large. Some authorities cite cold as a cause (though probably only of secondary importance) and condemn wet, draughty sties with a cold concrete floor. Gastro-intestinal catarrh may also be a contributory factor, owing to the diminished utilization of the lime-salts in food which it induces, and possibly in combination with an increased secretion

of lactic acid. The continuous and exclusive feeding on butter-milk has also been given as a cause among pigs. Certain breeds of pigs, and especially the finer ones, seem to inherit a predisposition to rickets, which since the introduction of the English breeds has increased. Concerning the action of infective matter in rickets nothing certain is at present known. Moussu is said to have induced rickets experimentally in animals (swine, goats) by inoculation with the bone-marrow of rachitic swine.

Post-mortem Conditions.—The chief pathological fact in rickets is that the bones remain soft owing to a defective deposit of lime, accompanied by peculiar processes of proliferation in the periosteum and the epiphyseal cartilage.

1. *The periosteum* is hyperæmic, showing upon its inner side a considerable proliferation and thickening of its bone-forming layer, in which process, however, the newly proliferated tissue does not calcify, but mostly remains soft. Not until later does this soft tissue ossify, when the bone appears thicker and stouter and shows localised hyperostoses. Such periosteal proliferations occur especially on the points of attachment of the muscles, for instance, in pigs, at the insertion of the psoas magnus and iliacus internus into the femur, and also often on the point of the os calcis. The thickened periosteum may become detached from some bones by the pull of the muscles, from the scapula, for instance, in pigs.

2. The chief changes in rickety bones show themselves on the epiphyseal border, and consist in an immense proliferation of the *epiphyseal cartilage* without proper calcification. Whereas normally the cartilage between epiphysis and diaphysis is separated from the latter by two thin parallel layers, the proliferation-layer and the ossification-layer, in rickety bones the former of these is excessively developed, while the latter is backward in growth. The two no longer run parallel, but are serrated and overlap. We find also in the proliferated cartilage-layer scattered foci of formation of medullary space and calcification: the formation of medullary space in rickety bones is consequently advanced beyond the line of ossification. This abnormal proliferation of the epiphyseal cartilage leads to the thickening and swelling of the epiphysis, which ossifies only during a prolonged course of the disease, and also to curvature and bending of the longer bones and to displacement of the epiphysis, from the connection between epiphysis and diaphysis becoming loosened.

From this proliferation of the cartilage and periosteum there arise numerous deformities in the bones. They are retarded in their growth (dwarfed growth), are short, broad and thickened, and show protuberances, especially near the joints (so-called double joints) and bend easily. Peculiar " badger-legged " or bow-legged curvatures occur on the extremities, lumpy elevations or large depressions upon the vertebral column (the upward deflections, causing " fish-back," are called cyphoses, and the downward ones, producing a sunken back, are lordoses, while the lateral curvatures are named scolioses). Upon the thorax a so-called pigeon-breast may develop, while the pelvis may become contracted in diameter. The bones themselves suffer infraction and fracture. The union of the ribs with the costal cartilage is frequently implicated; the abnormal proliferation of cartilage producing here the so-called rickety necklace (or rosary). Schütz also observed peculiar rickety changes on the skulls of dogs; whereas sound pups were born with their skull-bones completely closed, he found interstices, or fontanelles, between the several bones, these latter also being mostly attenuated. The joints likewise participate secondarily in these changes in the bones; for, consequent upon the softness and flexibility of the latter, the joints are more exposed to wrenchings and twistings than usual, so that rickets is often complicated with articular inflammation.

Symptoms.—The development of rickets is always gradual and slow, usually lasting several months. Its phenomena in the various domestic animals are mainly as follows

1. In **swine** a desire to lick things is said often to be a preliminary sign of rickets, though usually a stiff, constrained gait is the first to be observed (so-called " stiffness of sucking pigs "); the animals also arch their backs, lift the affected leg and can hardly be got to stand up. Bony tumefactions show themselves on the epiphyses near the joints, as well as on the ends of the ribs near the sternum, which are painful when touched. Further on appear curvatures in the bones; the feet are twisted forwards or backwards, the legs becoming bandy or bowed, and the astragalus often touches the ground. A sharp pressure upon the curved bones sometimes produces a crackling noise. The vertebral column is either bent forward (fish-back, cyphoses), or sinks downwards (hollow back, lordoses), or is bent sideways (scolioses); the pelvis is laterally contracted

upon the hip-joints, and may later become a cause of trouble in parturition (rickety pelvis). Occasionally we notice a simultaneous swelling of the nasal and jaw bones, as well as of the lower jaw, thus impeding both mastication and respiration; this condition represents what is called snuffling sickness. The animal's growth is consequently retarded and often remains dwarfed; the change of teeth is also gradually delayed. These curvatures of the bones are soon complicated with articular inflammation, with swellings and protuberances on the joints, especially on the ankle, carpal joint and pastern. As the disease advances appetite falls off, the animal grows very thin, has diarrhœa, becomes lame in the hind limbs and at last remains constantly lying. At times impetiginous skin-eruptions and bronchial catarrh may be perceived, and the animal finally perishes of cachexia.

2. In **dogs** rickets is manifested by bony tumefaction and especially in button-like thickenings on the border-line between the ribs and the costal cartilages (rickety necklace), curvature of the extremities (bandy legs, bow legs), chiefly of the radius and ulna forwards; in an unsteady, painful gait, placing the whole of the foot upon the ground, so that even the os calcis touches the floor; also in swellings upon the ends of the joints (double joints), development of a "pigeon-breast" (increased diameter from vertebral column to sternum and lessening of the transverse diameter of the thoracic cavity), etc. The change of teeth is often disturbed and retarded, the teeth are abnormally small and show defects of enamel. The dogs are also subject to eruptions.

3. In **horses** a stunted growth and fatigue on the slightest effort are first noticeable. The bulging of the bones occurs also, especially upon the epiphyses of the limb bones, particularly the shin-bone and bones of the head. Upon the latter they seem to be caused by the pressure of the halter and mostly upon the nasal bone. Swelling of the upper jaw is also not rare, and is produced probably by pressure of the molar teeth, which may even cause narrowing of the nasal orifice, difficulty of breathing and chronic nasal catarrh. The curvature of the leg-bones is usually not so great as in pigs and dogs; but we sometimes see foals whose forelegs are bent like those of a dachshund. Rickety foals often display a "goat-footed" appearance, knuckling over at the fetlocks and placing their hind feet

on the ground in a way resembling bears. The joints also are sometimes thickened, the hocks (a bulging of the bone like spavin) and the knees especially; and the bones evince great liability to fracture. The back is often either arched or sunk, and the process of dentition delayed. Now and then the horse manifests pain by shuffling about on its feet, twitching the hind legs or groaning. Finally, in foals, as in young pigs, rickets appears to cause a predisposition to eczema, bronchial and intestinal catarrh, decubitus and swelling of the thyroid gland. In isolated cases, rachitic stiff-leg is observed even in older foals (myogenic contraction).

4. In **cattle** the phenomena of rickets consist, as in other animals, in thickening of the bones, especially of the carpal and tarsal, curvatures of the back and leg-bones (X-legs, knock-knees), rickety necklace, rickety pelvis, painful gait and persistent lying down.

5. Among **poultry,** according to Zürn, rickets occurs most frequently in fowls from three to six months old, but is rarer in pigeons, geese and ducks. Its symptoms are continual sitting, difficulty in walking, knotty swellings on the joint-ends of the extremities and wing-bones, softness, flexibility and even curvature of the bones, arching of the breast-bone, etc.; also in wasting away and in poverty of blood.

Prognosis.—The forecast in rickets, as also in osteomalacia, is favourable when it is possible to remove its causes. By rational treatment very advanced changes in the bones of animals still young may be repaired. Abnormal deformities, sunken backs, rickety pelvis or thoracic malformation are naturally past all cure. Less valuable animals, therefore, such as pigs, it is better in such cases to slaughter promptly, as such deformed creatures are later of little economic use.

Differential Diagnosis.—Rickets can be mistaken only for pyæmic arthritis and acute articular rheumatism. Apart from the bulging and bending of the bones, these two diseases are distinguished by their sudden occurrence, and rapid, febrile course; pyæmic articular inflammation by its suppurative character.

Therapeutics.—The chief remedy for rickets is a prompt and persevering administration of phosphorus. We prescribe

for older foals single doses of $\frac{1}{8}$–$\frac{3}{4}$ grain; for dogs $\frac{1}{200}$–$\frac{1}{30}$ grain of phosphorus dissolved in oil or in cod-liver oil. For horses we would take, say, phosphorus 4–8 grains; cod-liver oil, 10 oz., daily one tablespoonful in bran-mash. For dogs and pigs: phosphorus, $\frac{3}{4}$ grain; cod-liver oil, 10 oz., daily a tablespoonful (for small animals a teaspoonful). To poultry give the last named solution in drops. A secondary remedy consists in an increased supply of lime. Prepared bone-meal mixed with the food, either a tea or tablespoonful, according to the animal's size; and for dogs also calcium phosphate in daily doses of 8 to 75 grains have both proved themselves efficient. Change the food if possible, but avoid over-feeding, remove any gastro-intestinal catarrh that may be present, and improve the conditions of breeding. With pigs a more natural mode of life must above all things substitute their unhealthy sty-existence, and they must be let out into the air and sunlight as much as possible. According to Merz, the dwellers near Lake Ammer cure their rickety chickens by feeding them on fish. Not only is this food nitrogenous, but fish-bones also contain much lime.

BRAN DISEASE (KRÜSCH DISEASE).

The bone-disease of horses described under this name is among foals of essentially rickety origin, and as it is caused by an exclusive diet of bran, it most commonly appears among millers' horses. The earliest *symptoms* are digestive troubles, constipation, weariness, profuse sweating during work, etc. These are complicated with swellings of the bones, especially near the carpal and tarsal joints, with lameness and passing attacks of pain, in the head-bones, and particularly those of the nose and jaw; also with difficulty in the prehension of food and in swallowing with loosening of the teeth, etc. The animals finally perish of cachexia and anæmia. When the disease attacks the young, it cannot be distinguished from rickets. Bran is a food quite as unsuitable for foals as potatoes for young pigs, and an exclusive bran diet can have none but injurious consequences, apart from the fact that it contains much phosphoric acid. Pütz considered Krüsch sickness as a kind of chronic phosphorus-poisoning, corresponding to the phosphor-necrosis of the upper and lower jaws in man, and traced it to the great proportion of phosphoric acid in bran. This hypothesis is, however, quite untenable. It is known that only free phosphorus, and not its acid, exercises an inflammatory action upon the bony tissues. In older animals this disease is probably identical with osteomalacia. Marcone has recently pointed out that the disease among horses which is known as osteoporosis or "bran-sickness," is absolutely identical with the osteomalacia of man,

and should properly be called equine osteomalacia. He considers that the disease is an inflammatory process of the bones, originating in the Haversian canals (osteitis rarefaciens), and accompanied by an increased excretion of phosphates in the urine (phosphuria). Some supposed cases may really have been actinomycosis.

Snuffling Sickness.—Under this name several authors have described a disease of pigs which is evidently not of uniform character. Sometimes the primary ailment seems to be rickets, at others a chronic hæmorrhagic, suppurative nasal catarrh, possibly of infectious nature, and again tuberculosis, actinomycosis or sarcomatosis. This labelling of varied complaints with one name renders impossible any definite classification of snuffling sickness, and it would be better to drop the appellation. The characteristic aspects are an audible snuffling respiration, which, at first intermittent, becomes later continuous, a swelling of the upper jaw, thickening of the nose, as well as shortening and distortion of the snout.

1. In the *catarrhal form* we also perceive at first a sero-mucous, and later a bloody-purulent nasal discharge, with frequent bleeding from the nose, which latter temporarily relieves respiration. We find also conjunctivitis, cough, difficulty in taking food, retching and, when the course is prolonged, also emaciation and death from cachexia or suffocation. *Post-mortem* reveals purulent, hæmorrhagic inflammation of the nasal mucous membranes, with extraordinary distortion and extreme atrophy of the turbinate and ethmoid bones. Schneider seeks the origin of this catarrhal form of snuffling sickness in a congenital, rudimentary structure of the bones just named, which promotes accumulation of foreign matter in the nasal cavity and a consequent inflammation of its mucous membrane.

2. The *rickety form*, which alone really belongs to this chapter, is also marked by rickety affection of the extremities. Haubold always found inflammation of the joints, and Lafosse noted also all the symptoms of rickets. In foals, similar swellings of the nose-bones and their surroundings occur during the course of rickets, accompanied by difficulty of breathing. Respecting the other forms of snuffling sickness nothing positive is yet known, and further investigation is to be desired.

The *treatment* of the disease varies with the primary affection, but is usually unsuccessful, and early slaughter is therefore advisable.

Acute Infectious Osteomyelitis.—By this name a febrile, infectious disease of young people is known, which occurs in human beings through South Germany, Switzerland and on the north German coast, and in which the long bones (femur, tibia and, more rarely, the radius and ulna) show purulent and ichorous foci of inflammation. It is specially distinguished as a genuine osteomyelitis from the secondary osteomyelitis which develops in the course of other infectious diseases (typhus, scarlet fever, measles, pyæmia, tuberculosis, cystitis, etc.) and in poisoning. The genuine infective osteomyelitis of man is produced by certain bacteria, viz., staphylococcus pyogenes aureus and albus. Its occurrence in the horse has recently been proved by Fröhner and Kärnbach. The condition is of considerable surgical importance.

CHAPTER XII.

TRICHINOSIS IN SWINE.

Natural History Notes.—The trichina (trichina spiralis) is a small thread-shaped round worm of the family of hair-worms (trichinidæ), which was imported into Europe during the thirties of last century, probably by Chinese pigs, and quickly spread all over the world. Paget discovered, and Owen first described and named, the muscle-trichina in 1835, Leidy found it in pigs in 1847, Leuckart and Virchow proved the presence of intestinal trichinæ, and Zenker demonstrated the trichinosis of man in 1860, in Dresden. Two development forms of the trichina must be distinguished: the sexually ripe intestinal trichina and the sexless muscle-trichina.

1. *The intestinal trichina* lives on the superficial layer of the mucous membrane of the small intestine, is straight and has a pointed head. The male is $\frac{1}{20}$ inch long and the female $\frac{1}{8}$ to $\frac{1}{6}$ inch. Its digestive canal consists of mouth, œsophagus, stomach, intestine, anus and a cleft-like cloaca; the generative organs of testicular-tube, spermatic duct and male sexual opening on the one hand, and upon the other of the female ovary, uterus, vagina and vulva. A female produces 1,500, according to Leuckart, or 10,000, according to Braun, living young during an average life of from five to six weeks.

2. *The muscle-trichina* represents the larval form of the above, from whose embryos it develops. Its length is up to $\frac{1}{25}$ inch; it possesses a pointed head and a round, cloven hinder part (cloaca). It lies in the muscles in oval to round cysts, from one to four in each, rolled up in serpentine, spiral or annular form, or in the shape of a pair of handcuffs. The life-duration of a trichina may, in a pig, amount to 11 years (Dammann) and in a man even to 31 years.

The **Development of Trichinæ**—according to the investigations of Leuckart, Zenker, Pagenstecher, Haubner,

Virchow, Graham, and others—is as follows. The starting point is the consumption of muscle-flesh containing trichinæ. It is still uncertain whether embryos of intestinal trichinæ evacuated with the fæces are directly ingested. Attempts to inoculate by feeding with intestinal trichinæ have but rarely succeeded. On the other hand, it is not impossible that undigested morsels of flesh containing them may be passed with the dung, which, on being consumed by other animals, cause trichinosis; in this case again the muscle-trichina, and not the dung, is the infecting medium. We can distinguish four stages of development: 1. The *stage of embryo-formation in the intestine*, beginning on the seventh day after their reception. The encysted muscle-trichinæ are in 24 hours set free by dissolution of their surrounding capsule; in another 24 to 48 hours they are sexually ripe, copulate, and bring forth living embryos on the seventh day, reckoning from that on which the meat was eaten. Both males and females then bore their way into the crypts of Lieberkühn, where the females deposit their eggs (Graham). The females are found under the intestinal mucous, in the bowel wall, and even in the mesentery itself, where they make their way in order to deposit their young (Heitzmann). 2. *The stage of immigration of the embryos into the muscles*, which lasts from two to three weeks. From the seventh day forward the embryos begin to perforate the walls of the intestine in which they were born and to work their way into the muscles, partly through the vascular system and partly along the connective tissues, the duration of such emigration being from nine to ten days. As Graham has recently shown, the larvæ quit the intestine exclusively by way of the lacteals of the intestinal villi. They are then carried automatically, by way of the thoracic duct, into the blood-stream and from thence, also mechanically, into the muscular tissue itself. 3. *The stage of encapsulation in the muscles*, lasting from four weeks to three months. On reaching the muscles the embryos lie dormant for about sixteen days and then change into muscle-trichinæ. The muscular fibres lose their diagonal striation, the sarcolemma tube becomes dilated like a spindle around the embryos which have penetrated into the middle of the fibres; the muscle-nuclei multiply, while the contents disappear, the connective tissue is infiltrated with small cells, and the capillaries are much distended. Finally, regular layers of cells form round the trichinæ, which thicken into a round, lemon or eye-shaped cyst of connective tissue, whose two pointed ends are

darkened at the poles. 4. The *calcification stage of the cysts*, lasting from three to eighteen months. After the third month the cysts begin to calcify, so that the trichinæ themselves can no longer be seen. The calcified cyst can then be perceived by the naked eye on a thin section as a small light spot, about $\frac{1}{25}$ inch long. The trichinæ often live for a long time inside the calcified cysts without themselves undergoing calcification. Trichinæ have been found in the adipose tissue (bacon-sides) as well as in the muscles, both in their free and their encysted condition (Chatin). The embryos of trichinæ have also been observed in the blood, the blood-vessels, the lungs, the lymph-glands, etc. As a rule, however, the larvæ only attain to complete development and to the formation of cysts in striated muscles furnished with a sarcolemma. In other organs they invariably perish.

Occurrence.—Up to the present trichinæ have been observed in man, swine, rats, mice, dogs and cats; also in the wild boar, fox, bear, marten, pole-cat, domestic fowl, turkey and jay. They have, moreover, been brought to development by artificial feeding in many of the mammalia, such as the horse, ox, sheep, dog, rabbit, ferret, hare, raccoon, badger and guinea-pig. Their favourite muscles are the crura and the muscular part of the diaphragm, the tongue, muscles of the larynx, masseters, those of the eye, the temporal and intercostal muscles, also those of the neck and trunk and the twin or gemelli muscles. So far no muscle-trichina have been discovered in the heart or smooth muscles, but they have been found in the adipose tissue near muscles. The number of individual trichinæ in the different muscles varies greatly; as many as 4,000 may be present in 15½ grs. of flesh. The total number of trichinæ in a swine was estimated in one instance at sixty millions.

The *statistics* of the spread of trichinosis among pigs vary greatly in different countries, but the disease is generally more common in north than in south Germany. In Prussia about one pig in every 2,000 seems to have trichinosis according to the figures given, but in Germany generally the proportion is only one in 10,000. In America, on the contrary, the percentage is much higher, the investigations of Billings (in Boston) showing one in twenty-five, or 4 per cent. Concerning the ham and other swine-flesh imported into Germany from America, we may assume as the result of present researches that an

average of 2 to 3 per cent. contains trichinæ (a maximum of 8 per cent.).

Pathogenesis.—The manner in which swine are infected with trichinæ has, up to the present, not been satisfactorily explained. As the main source of infection trichinous rats and mice, which pigs greedily devour, are chiefly suspected, and such trichinous rats are mostly met with in horse-slaughtering establishments and abattoirs. In eighteen horse-slaughter houses Leisering discovered fourteen infested with trichinous rats. Franck found 9 per cent. of rats brought from flaying-houses to be trichinous, and Huller 8 per cent. of those gathered from various places, thus indicating that a connection between rats and swine is beyond all doubt. Pigs may also be directly infected by feeding them with the flesh of other pigs which have died of the disease, or with the offal and slaughter-house refuse of other infected animals; possibly also indirectly by taking up the dung of swine containing undigested morsels of trichinous flesh. According to Hertwig's observations it seems that successive immigrations of trichinæ may occur, as they are found in various stages of development in the muscles. We must await further researches on the possibility of infection by the embryos of intestinal trichinæ passed out with the fæces. The occurrence of congenital trichinosis is more than doubtful.

Symptoms.—Apart from the cases of experimental feeding, no symptoms of trichinosis have yet been observed in swine during life, probably because such symptoms, being so little characteristic, have been overlooked by owners during the period of immigration, or confused with other diseases. Later, when the immigration is ended, the general condition of the trichinous animal is in no way disturbed. Usually when the pigs are killed the time of immigration is long since over, which explains the fact that, in spite of the proved presence of great quantities of trichinæ in the muscles, no signs of illness could be perceived immediately before slaughtering. Moreover, the absorption of a small number of trichinæ causes no morbid symptoms. The aspect of trichinosis, as produced experimentally in pigs by feeding them on trichinous flesh, allows us to distinguish two stages, that of an intestinal affection, as well as the subsequent derangements in the muscles.

1. The *affection of the intestinal canal* occurs at the end of the first and beginning of the second week after infection.

It betrays itself by loss of appetite and spirit, in arching the back, tension of the abdomen, continued diarrhœa with signs of colic and much debility; in a few cases vomiting was also noticed. In young animals, or if the quantity of trichinæ absorbed be very large, the disease may prove fatal even in this stage. On the other hand, if such quantity be only moderate, the animal may recover.

2. The *symptoms* are observed in connection *with the muscles* with the immigration of the embryos into their fibrils, and the development of inflammatory conditions; they occur therefore in the second and third weeks. The animals display a peculiar itching, which makes them rub and scratch themselves; also a stiff and constrained attitude of the limbs, which may amount to lameness and weakness; they manifest pain, rapidly lose flesh and constantly lie down. Difficult mastication and swallowing are also observed, laboured and painful breathing, a feeble, hoarse voice, and also œdematous swellings in various parts of the body. Usually a complete recovery is made after the illness has lasted from four to six weeks, when the animals quickly grow fat again. In rare cases only is the result fatal.

Trichinosis in Dogs.—A one-year-old dog which was fed on very trichinous pork manifested signs of restlessness and bloody diarrhœa after two weeks, and a little later symptoms of lameness and spasms. The neck-muscles especially were attacked by tonic-clonic spasms, which sometimes spread over the whole body. The hind legs were completely paralysed, and the temperature rose to 103° Fahr. On dissection, numerous migratory trichinæ were found in the muscles. Another very old dog remained quite healthy in spite of the same food (Dlugay). Three dogs fed on trichinous meat showed symptoms of diarrhœa and lassitude and upon *post-mortem* examination, six weeks later, numerous encysted trichinæ were found in their flesh. Two cats, which formed the subject of experiment, died of intestinal trichinosis (Leistikow). Perroncito's experiments give similar results. Pirl induced trichinosis of the muscles in guinea-pigs by feeding them upon infected dog's flesh. In the Chemnitz slaughter-houses in 1897–8, 1.4 to 1.3 per cent. of all dogs were trichinous (Tempel and others). In Saxony in 1900, 0.4 per cent. of all the dogs examined were trichinous.

Diagnosis.—It is impossible, as a rule, to recognise trichinosis during life, its symptoms being so little characteristic and allied to those of intestinal catarrh or muscular rheumatism, and because the trichinæ, once encapsuled, cause no further morbid disturbances. The demonstration of the disease is easy after slaughter; for even with the naked eye the calcified cysts can be detected in thin slices of the flesh as fine,

light-coloured spots. A positive diagnosis is, however, possible only by the aid of a microscope, for which an enlargement of forty times is most suitable. The simple method employed is to clip thin sections with sharp, curved scissors out of the muscles most frequently attacked, and, if possible, close to their place of insertion, parallel to the line of their fibres. Place these on the glass plate which serves as object-bearer, spread them out and cover with a similar glass, squeezing them so as to be transparent. According to the size of these glass plates, a number of sections may be examined at once. To ensure accuracy in the results, the German law since 1900 has required that in the case of the whole swine, at least four morsels of flesh of the size of a bean or a hazel-nut should be cut from different muscles (crura of the diaphragm, rib portion of the diaphragm, tongue and larynx muscles). Of each of these, six specimens of the size of a grain of oats are to be prepared and examined. In the case of preserved meat (ham, bacon, pickled meat), specimens the size of a grain of oats are to be prepared from three portions of the flesh, free from fat: four specimens of bacon and six specimens of ham.

Differential Diagnosis.—Microscopical examination of the muscles for trichinæ sometimes leads to confusion with other and similar formations in the muscles. Among these are first the so-called concretions. These are greyish-white bodies, about the size of a millet-seed, which often permeate the muscles in large numbers and apparently vary greatly in their nature. Sometimes they consist of crystals of lime, tyrosin, margarin and stearin, at others they appear as calcified cysticerciæ or new growths, and were formerly supposed to be concrements of guanin. They all differ from trichinæ in being considerably larger, in dissimilarity of shape and in the absence of a round-worm in their interior. In particular they lack the eye-shape of the trichinæ-capsule, its darker poles and sharp contours. Trichinæ may also be mistaken for Miescher's sheaths (or tubes); but these possess no capsule, and are longer in shape with blunt ends, are tightened up into sections (like sausages), are mostly also larger and consist of dark, granular matter, which, under a more powerful glass, can be seen to be crescent-shaped bodies; finally, the diagonal striation of the muscular fibres immediately surrounding these Miescher's sheaths remains quite unimpaired. Trichinæ have been confounded with the spores of actinomyces (Dunker), which occur in the form of round or bean-

CHAPTER XIII.

"MEASLES" OF DOMESTIC ANIMALS.

I.—MEASLES IN PIGS.

Natural History Notes.—This disease, which was formerly known as miliary fever or pearl-rash, is produced by the bladder-tail parasite of the connective tissue, or gargol (*cysticercus cellulosæ*), which is the larval form of the tænia solium of man. The connection between the hermit tape-worm of man and the cysticercus cellulosæ was first established by Küchenmeister and Haubner by means of feeding experiments. Measles, therefore, in pigs depends in its frequency upon the occurrence of tænia solium in man. The swine are infected by absorption of human excrement containing proglottides, and owing to their preference for such excrement, pigs are of all domestic animals most liable to this form of infection. Those which feed in the open and on fields largely manured with refuse from city middens and cesspools, or which live in dirty sties situated near human privies, most frequently suffer. The disease consequently varies much in frequency according to the manner in which swine are kept.

The development of cysticercus cellulosæ takes place when the eggs of tænia solium pass into the stomach with the proglottides—which latter may already be in a putrid condition—and are there digested by the gastric juices. The embryo (which has six hooks) is thus set free, bores through the intestinal wall and wanders on, partly along the connective tissue, and also as it is carried by the blood. These immigrations, however, occur only with younger pigs, up to six months of age; older ones not being infected. When once the embryo is affixed, it loses its hooks, becomes bright in the centre, proliferates outwardly and changes into a vesicle, upon whose inner wall a peg-shaped body grows, which is the rudiment of the scolex. In twenty days the parasite is the size of a pin-head, the future

head being indicated by a dull dot ; in forty days it has grown as big as a mustard-seed, the head being distinctly seen, though the suction-cups and hooks are still in the rudimentary stage. At the end of sixty days it has attained the size of a pea, hooks and suction-cups are clearly visible, but there is no neck ; after three months the cysticercus is fully developed and able to infect mankind. Round its location a reactive inflammatory process is set up, which leads to the construction of a capsule of connective tissue. It must not be overlooked that among those forming the same immigration much variety of development occurs, some cysticerci dying early and becoming calcified, while the rest attain perfect growth. According to Ostertag's observations, those which find their way to the liver and lungs usually die off soon. In addition to swine, the cysticercus cellulosæ is found in man, the dog, the ape, the bear, and also in sheep, deer, cats and rats.

Occurrence.—The favourite localities for cysticercus cellulosæ are the interstitial connective tissues of the muscles. Of these the diaphragm, loins, tongue, heart, neck, muscles of mastication, intercostal muscles, adductor muscle of the lower leg, and the muscles of the thorax are most frequently affected (Ostertag). The cysticerci also occur in the brain, in the fat, and in the lymph-glands situated in the fat ; while they are found but rarely in the lungs, liver, spleen and eye. Sometimes all the body organs are inter-penetrated by thousands of these parasites, so that there may be from three to four for every scruple of flesh. They are rarely isolated. The fully-developed cysticercus varies in size from a pea to a bean, and presents the appearance of a bladder of dull white colour and semi-solid consistence, which has both a head and neck. The former is an opaque dot, usually curved inwards, giving the bladder somewhat the form of a kidney, whereas, when the head is convex, it has more the shape of a round-bellied bottle. Upon the head are four suction discs, a rostellum and a double circlet of hooks with twenty-six to thirty-two hooks. The bladder is usually enclosed in a capsule of connective tissue, which is absent only when in the serous body cavities. These vesicles swell out when boiled and creak on being cut ; those that have died undergo fatty degeneration and calcification. The muscles round about remain at first normal, but later, as the cysticerciæ grow numerous, become greasy and watery.

The frequency of measles varies much in different districts.

In consequence of the strict enforcement during the last ten years of the regulations relating to the inspection of meat, measles occurs but rarely in home-grown German swine, and in South Germany it may be said to have disappeared altogether. On the other hand, the disease is very rife among swine imported from Servia, Russian Poland, and Galicia (50 per cent.). Statistics show that in Prussia, for a period extending over seven years (1876–1882), and including a total of twenty millions of swine, the proportion of measles was 3 in 1,000. The aggregate of the years 1890–93 gave similar results; among 5½ millions of swine slaughtered, there were 14,000 cases of measles. Recent statistics show that the conditions have considerably improved. In 1896–98 the percentage of measles was 0.7 ($\frac{7}{10}$) annually. In 1899–1901, 29 millions of swine were examined and the proportion of measles was only 0.5 (½) *per thousand.* In Prussia at this present time the cases of measles are 1 in 2,000. Twice as many occur in East as in West Prussia.

Symptoms.—These are by no means characteristic. Many pigs, although highly infected, reveal no morbid symptoms whatever during life. The following are usually cited as indications of the disease. A hoarse, rough, croaking grunt, falling out of the bristles, faintness and depression, lessened appetite, paleness of the oral mucous membrane, anæmia and emaciation, œdematous swelling of the head, neck, etc., diarrhœa, weakness, paralysis and finally death from exhaustion. In a few cases sudden death occurs, when, for example, an unusual quantity of the tape-worm eggs has been absorbed. The aspect of the disease is much more clearly marked when localised upon any special organ of importance. Thus we may observe, after immigration of the parasite into the brain, severe cerebral phenomena in the form of epileptiform spasms, paralysis of one or both sides, mania and raving. Or if it attain the eye (beneath the retina, in the vitreous humour, the lens or the chamber of the eye), we notice blindness. When the parasites are too numerous in the tongue, that member becomes paralysed, and the animal cannot take its food; excessive localisation upon the serous membranes causes peritonitis and pleuritis. Finally, sudden and apoplectic death has been known to occur.

Diagnosis.—The identification of measles in swine is possible only during life when the cysticerciæ can be felt or

seen on the under side of the tongue near its frænum, or upon the inner surface of the eyelids, where they appear through the mucous membranes as hard nodules. It is recommended, when the former is not the case, to make an incision lengthwise in the tongue in order to lay bare any that may be present. It is possible that in future the ophthalmoscope may be of service in this diagnosis. We cannot, however, conclude from the absence of the cysticerciæ in the tongue that the animal is entirely free; for out of forty-one diseased animals Railliet found the parasite there in only thirty-one cases.

The cysticerciæ cellulosæ are easily recognised in the slaughtered carcase, difficulty only arising when they are already dead and degenerated. Diagnosis is in all cases assured by microscopical identification of the head, hooks or external case. Ostertag's investigations have established, with regard to the diagnosis of dead cysticerciæ, that the discovery of so-called lime-corpuscles in a calcified or caseous muscle-focus is a positive proof that such focus has been produced by a bladder-worm. To certify the presence of cysticerciæ in minced or sausage-meat Schmidt-Mülheim recommends, in preference to the tedious microscopical method, that such flesh be digested for several hours in six to eight times its weight of artificial gastric juice at 104° Fahr., whereby all but the head and its circlets of prehensile hooks is digested, and these, gathering at the bottom of the glass, can then easily be detected by a powerful lens. This artificial gastric juice is prepared by pouring glycerine over the chopped mucous membrane of a pig's stomach and allowing it to act for some time, when a small quantity of this pepsine-glycerine is replaced by a ½ per cent. solution of hydrochloric acid. Falkone has repeatedly observed instances of cysticercus pisiformis as large as a pigeon's egg in swine.

Prophylaxis.—The precautions to be adopted against measles in pigs, consist in preventing them from consuming human fæces, cleansing and improving their sties, avoidance of pasture-feeding, of human privies and dung-heaps. This refers only to the young pigs up to six months old. To this should be added, if possible, the treatment and cure of all human beings suffering from tape-worms and extirpation of the latter. No treatment of the sick pigs themselves is of any avail. According to German law, measles requires a period of warranty of fourteen days.

Man is only **infected** by measly pork when it is eaten raw or half raw, and in the latter connection half-cooked sausages are particularly dangerous. The prophylaxis is in such cases very simple, as with trichinosis, and consists in thoroughly boiling or roasting the meat through. According to Perroncito's tests, the cysticerciæ perish if exposed longer than a minute to a heat of 122° Fahr. They are also killed by being well smoked, pickled or dried. As a result of their loss of water they shrivel up to the size of a pin-head, and become hard bodies which crackle between the teeth. The legal enactments concerning the consumption of such flesh vary in different countries; but its use ought to be strictly prohibited, or restricted to industrial processes, such as soap or glue-manufacture. This prohibition should, in our opinion, extend also to the fat and bacon, which are sometimes infected, and which people often consume raw. On the other hand, meat only slightly measly (*i.e.*, containing only solitary cysticerciæ) might be admitted to consumption under certain restrictions. These should consist in boiling under proper supervision, and only allowing it to be sold after duly informing the buyer of its nature and how it should be treated. The dangers arising in man from eating measly pork are, first, the incorporation of one or more tape-worms, which may cause disturbances of digestion and nutrition and lead also to psychical affections; also the self-infection, through the eggs of tænia solium, with a formation of cysticerciæ in the brain, eyes, etc., producing functional disturbances of the brain, blindness or even sudden death. In consequence of the care exercised in inspecting meat in our larger cities, tænia solium in man has become very rare. In Berlin, for instance, the common tape-worm is tænia inermis.

II.—MEASLES IN CATTLE.

Natural History Notes.—The measles of cattle represent the sexless larval stage of tænia medio-canellata (saginata, inermis) of man. It is rarer than swine-measles, but more common than is generally supposed. Its development also depends on the consumption of the proglottides and eggs discharged with human excrement. In by far the larger number of cases the parasite infests the inner and outer masticatory-muscles, and in a secondary degree the heart, the proportions being about 9 to 1. Occasionally the tongue, the muscles of the neck and breast, and the adipose tissue are attacked; but very rarely the liver, lungs, brain, kidneys, etc. A further distinction between the disease in cattle and in pigs is that the parasites are not so numerous as the cysticercus cellulosæ. Usually only a solitary one is found in the muscles of the jaw. In Berlin, among 1,900 cattle with measles, the cysticercus was found singly in 1,825 (95 per cent.). Occasionally, however, the parasites are distributed over the entire

body. In one case Hering counted 300 in half a pound of ox-flesh. Of 389 measly cattle examined, the parasite was only in 22 cases found to be spread through the whole muscular apparatus of the body. Zenker and Railliet have also discovered it in the goat.

The development of the parasite in cattle is essentially the same as in swine. Microscopical examination shows that it has no rostellum and no circlet of hooks, hence its name of tænia inermis, or unarmed tape-worm. The four suction-cups are strongly developed, and on the crown of the head is also a frontal suction-disc. Hertwig's tests show that its organic development is ended in about 18 weeks, and its growth in 28 or a little more. Here also dead parasites are found with living ones. In 30 days they are $\frac{1}{8}$ inch long and $\frac{1}{7}$ inch broad. The head appears as a grey point, one hundred and twenty-fifth of an inch in diameter. The scolex shows 4 sucking and 1 central orifice, which appear as flat discs. At the end of 60 days, the cysticercus has attained a length of four twenty-fifths of an inch and the head is as large as that of a pin. At the end of 90 days it is one to six twenty-fifths of an inch long and four twenty-fifths wide. After 130 days its length is seven twenty-fifths, and after 160 days nine twenty-fifths of an inch. By this time a small central orifice has become distinctly visible between the four sucking discs, the scolex is as large as a poppy-seed, one hundredth of an inch long and seven-hundredths of an inch wide. Older cysticerci may grow to a length of twelve twenty-fifths of an inch in length, with a scolex as large as a grain of mustard-seed.

Occurrence.—Measles in cattle is of more frequent occurrence than was formerly believed to be the case. At any rate, the tape-worm whence these spring is much commoner in man than that which causes swine-measles, the latter having been almost extirpated in large towns by strict supervision and wise hygienic measures. Improved methods of examination have shown the cattle-parasite to be much commoner than was believed. Although in former years only isolated cases of measles in cattle were reported in Berlin, since the discovery of the favourite seat of the parasite the number of cases has risen from 60 in 1888, to 112 in 1887 and to 389 in 1890; 1,623 cases of measles were reported in Berlin in the course of five years (1892–1897). In Prussia the numbers were as follows: In 1894, 748; in 1896, 1,918; in 1897, 2,629; in 1898 and 1899, 0.5

(½) per cent.; and in 1900, 0.6 (three-fifths) per cent. (7,000 animals) of all the cattle slaughtered in Prussia were found to be suffering from measles. Bulls are more frequently attacked than cows. The disease is more or less prevalent in other countries (Switzerland, France, Hungary and Italy). Its symptoms occur with special frequency in Italy among slaughtered cattle. According to Alix, one-fifth of the cattle slaughtered in Tunisia show its presence, which he explains on the ground that the beasts are only half-fed and therefore consume dung-smeared fodder which better-fed cattle would despise, and thus become infected with the eggs of the human tapeworm.

Symptoms.—The disease occurs in cattle without any symptoms. Even in the special feeding experiments no marked signs were noticeable in many cases (Gerlach, Perroncito, Pütz). Leuckart, Mosler and Zürn, on the contrary, succeeded in producing a decidedly morbid aspect in a part of their trials on calves, to which they gave the badly chosen name of "acute cestoid tuberculosis," because its peculiar sectional result reminded them of tuberculosis. These phenomena consisted in rise of temperature, diminished appetite, signs of pain on palpation of the hind quarters, groaning, staggering, stiff and painful gait, increasing emaciation and debility, inability to rise, constant lying, difficult breathing, diarrhœa, fall of bodily temperature and death on the twenty-third day. On dissection, thousands of the parasites were found in the heart, midriff and in the muscles of jaws, tongue, neck and belly. Ostertag has proved by experiment that a complete spontaneous recovery from measles is possible in the case of cattle. This would account for the fact that cysticerci are found far less frequently among older cattle than among young animals (arrested development of the parasites).

Diagnosis.—It is usually impossible to recognise the measles of cattle during life, unless, as in pigs, the presence of the parasite should be discovered under the tongue. It is said to be the rule in Southern France to examine the tongues of slaughtered cattle, as is done in swine for this purpose. This invasion of the tongue by the parasite may, however, lead to a confusion with antinomycosis, as Noack has pointed out. Moreover, its discovery is much more difficult than is the case in pigs, and probably it has hitherto been generally overlooked.

The frequency of tænia mediocanellata in man is out of all proportion to the discovery of cattle-measles in the slaughter-houses. This difficulty of demonstration arises partly from the fact that most of the parasites appear singly, and partly because, on contact of the flesh with air, they soon become invisible, also because they often lodge in the adipose tissue. The experience of the Berlin and Magdeburg abattoirs proves that their favourite haunts are the inner and outer muscles of mastication. Glage's observations show that by cutting into the outer muscles of mastication the presence of cysticerci, which would otherwise have passed unnoticed, may be frequently proved.

Differential Diagnosis.—The parasite may be mistaken for the *cysticercus tenuicollis*, which is only found under the serous membranes and in the liver, and which is furnished with a double row of hooks ; the *echinococcus*, which is round in shape, without scolex, and has a laminated epithelium and *lymph-cysts*.

Prophylaxis.—As in pigs, this consists in preventing all consumption of the proglottides and eggs passed with human fæces, but is much more difficult. The staff employed in the byres should, however, be strictly prohibited from evacuating therein.

Human infection occurs solely through eating raw or half-raw beef, especially under-done beefsteaks. The best protection then is thorough cooking through, as the parasites are destroyed by a temperature of 112° Fahr. (45° C.). Other prophylactic measures are pickling, freezing, and keeping the meat for three weeks in a cold chamber in order that the parasites may perish spontaneously (Ostertag). The tape-worm of cattle-measles is much more difficult to drive out from the human body than tænia solium. As regards meat-inspection, measly beef should be treated exactly the same as infected pork. In Prussia, if a single parasite be discovered, and that only in the jaw-muscles, the whole carcase is condemned as injurious and may only be sold for domestic use after being boiled under police supervision. (Cp., pp. 616 and 622, slaughter enactment regarding infected swine's flesh.)

III.—MEASLES IN DOGS.

In isolated cases the larva of tænia solium has been found in the brain, muscles, liver, lungs, sub-cutis, etc., of dogs, and the infection must have come from man. Usually the symptoms were those of cerebral affection : mania, epileptiform spasms,

rotary movements and singularly sudden death. More rarely there were signs of muscular disorder. Thus in one case Trasbôt noticed absolute incapacity for movement, with great pain when touched or when the limbs were stirred. Gambarotta saw a dog die of weakness in which all the organs, brain and spinal-cord were infested with cysticerci. Sub-cutaneous cysticerci have been observed in the dog by Railliet, von Rátz, Meyerstrass and others. Noack asserts that he also found the cysticercus tenuicollis in the brain of a dog. Avéradère observed a cysticercosis of the pleura and peritoneum, accompanied by peritonitis, in lambs, which was due to the presence of cysticercus tenuicollis.

CHAPTER XIV.

MIESCHER'S TUBES.

(*Sarcocystis Miescheriana.*)

Natural History Notes.—Our knowledge even to-day is very insufficient respecting the nature of the tubes discovered in 1843 by Miescher in the muscles, and which were to some extent re-discovered by Hessling in 1854 and by Rainey in 1857. These Miescher's tubes are formations of spindle, roller or thread, shape in the interior of the primitive fasciculi of the muscles, whereby the latter are made to bulge out. They are microscopically small, sometimes visible as fine, white, elongated lines, especially in pigs; and sometimes attain a length of $\frac{3}{4}$ inch, as may be seen in the larger animals. They are mostly disseminated, although present in very large numbers, but agglomerations are occasionally found as large as a pea or a hazel-nut. They are externally bounded by a thick cuticula. Within the tubes are an extraordinary number of minute objects (germs), shaped like sickles, kidneys or beans, which lie together in a homogeneous fundamental substance grouped into divisions like the meshes of a net, by which the larger tubes are constricted like pouches. On puncturing the tubes, a milky, purulent mass oozes forth, containing millions of sickle-shaped germs, which may often be entirely squeezed out, so that only the empty husk remains.

These objects were at first regarded as fungi, afterwards as accumulations of lymph-cells, and subsequently as a stage in the development of swine-measles; later investigation has, however, proved them to be sporozoa, which live as parasitic colonies surrounded by a tube in the diagonally striated muscles (so-called sarcosporidiæ). While the tubes remain closed, they appear to be harmless, but if these burst, the sickle-shaped spores within are changed into amœboid migratory cells, which penetrate the neighbouring muscles as an inflammatory infiltra-

tion (myositis sarcosporidica s. gregarinosa) and produce a specific muscular disease with atrophy of the muscle cells and formation of tumescent inflammation-foci (white or grey sarcosporidial tumours in the shoulder and loin muscles of horses, which are hard and as large as the fist). In sheep the early rupture of these tubes causes roundish, encapsuled tumours the size of a pea, which penetrate towards the free upper surface of the pharynx and pleura (sarcosporidial cysts).

Blanchard has set forth the following system: Class: Sporozoa; Order: Sarcosporidiæ. I. Family mischeridæ, parasites in the striated muscles with two varieties: 1st—genus mischeria (thin membrane, homogeneous); 2nd—genus sarcocystis (thick membrane, permeated by fine caniculi). II. Family balbianidæ, parasitic in the connective tissue (thin outer membrane, homogeneous), with one species: genus balbiania. Miescher's tubes (sarcocystis miescheriana) belong to the mischeridæ, and the so-called psorospermic cysts of ruminants (balbiania gigantea) to the genus balbiania. These occur oftenest in the interstitial connective tissue of the pharyngeal muscular apparatus of sheep and goats. (Morat found 30.2 per cent. out of 900 pigs to have psoro-spermian nodules in the pharynx.) The genus mischeria is commonest in our domestic animals, especially in pigs. Ripping asserts that Miescher's tubes were not absent from any pigs examined by him; Kühn noted them in 98.5 per cent. of all animals he examined; and Koch in 8 per cent. of all pigs slaughtered in Vienna. Next in frequency of attack come horses and cattle.

Occurrence.—Miescher's tubes are only found in the muscles of herbivora and omnivora, especially in pigs, the ruminants (ox, sheep, deer) and in horses. They have also been met with in buffaloes, reindeer, goats, hares, rats and mice, in fowls and other birds, but never yet in the carnivora. The largest tubes (sarcosporidiæ gigantea) occur in goats, sheep and buffaloes. They are most frequently found in the muscles around the cavities of the mouth and throat—for instance, in the œsophagus, tongue and muscles of the jaws, cheeks, larynx and pharynx; although often numerous in other muscles, such as the neck, loins, abdominal walls, thighs, midriff, heart, etc. Sometimes they lead to interstitial myositis with atrophy and cellular infiltration of the connective tissue of the surrounding muscular fibres. In other cases the flesh—*e.g.*, that of full-grown cattle—assumes a pale colour like that of veal, owing to the presence in large quantities of sarcosporidiæ. In older horses and in buffaloes they seem to be almost invariably found; also in pigs Perroncito discovered them in a quarter of those examined. Among 100 cachectic sheep examined by Moulé, 99 had sarcosporidiæ.

Symptoms.—Although Miescher's tubes frequently occur in the muscles of herbivorous domestic animals, they rarely cause any signs of illness in their hosts; only in solitary cases have any pathological symptoms been observed which arose from muscular changes of inflammatory nature occasioned by an excessive number of these tubes (myositis sarcosporidica). Thus Dammann and von Niederhäusern noticed difficulty of breathing in a sheep and a goat caused by inflammation in the muscles of the larynx and pharynx; Brouwier and Tokarenko observed lameness in a bull and difficulty in rising, and, indeed, complete muscular paralysis; Virchow, paralysis of the hinder limbs in pigs. Brschosnionski noticed loss of appetite in two pigs, continual lying, arching of the back, irregular movement of the hinder parts, pain in the muscles when pressed, hoarseness and fever (trichinosis at first suspected); after slaughtering, the muscles were found to be watery, opaque and permeated by numerous sarcosporidiæ. Höflich describes a case in a horse of glossitis sarcosporidica which terminated fatally. Hendrickx and Liénaux found sarcosporides in the hard knotted swelling of the tongue and upper lip of a horse.

Anatomical changes are more frequently found in the flesh of slaughtered animals. According to Hertwig, Miescher's tubes were so numerous in six pigs that the muscular preparations contained as much of tubular as of fibrous matter. The flesh was consequently flabby, extremely watery, and its section-surface assumed after a few hours a pale green colour. For differential diagnosis, consult the chapter on Trichinosis.

Rieck believes that, although sarcosporides do not as a rule give rise to any conspicuous pathological changes in the muscular structure, they may, under certain conditions, set up an interstitial myositis, at first acute, and subsequently passing into the chronic form, and accompanied by secondary degeneration of the muscular fibres. The condition is brought about by the influx of the embryos into the interstitial connective tissue of the muscles. With the penetration of the grain-like bodies into the muscle fibres, where they form cysts, the disease passes into the second or chronic stage in which the exudation from the cells becomes changed into fibrillary connective tissue. Siedamgrotsky, Laulainé and Brouwier are of opinion that the chronic interstitial inflammation which occurs in horses, cattle and swine is due to the presence of sarcosporides, and this view is confirmed by Pfeiffer, Rieck and Höflich. Pütz, Eberth and Schmidt-Mülheim, however, hold contrary opinions.

CHAPTER XV.

DISEASES OF THE NERVOUS SYSTEM

I.—DISEASES OF THE BRAIN AND ITS MEMBRANES.

Inflammation of the Brain. Meningitis and Encephalitis.

General Notes on Inflammation of the Brain.—The inflammatory affections of the brain may be classed according to their anatomical aspects as follows :—

1. *Pachy-meningitis, or inflammation of the dura mater.*
2. *Lepto-meningitis, inflammation of the pia mater.*
3. *Encephalitis, or inflammation of the cerebral substance itself,* including *brain-abscesses* and also some *softenings of the brain.*

Lepto-meningitis may be either serous, suppurative, croupous or tuberculous in type. We also speak of a convexity-meningitis and a basilar-meningitis, according to the seat of the inflammation. A simultaneous affection of the meninges and of the brain is named an encephalo-meningitis. This exact, anatomical classification, excellent as it is, cannot, however, be adopted in a clinical description of these various diseases in domestic animals. The conditions are very different from those of human pathology. A clinical differentiation between meningitis and encephalitis in domestic animals is extremely difficult, if not impossible; for they present essentially the same symptoms, and also when the membranes are inflamed the brain itself is generally implicated. It is certain that what is classed as inflammation of the brain in our veterinary medicine is in by far the greater number of cases lepto-meningitis. Positive proof, however, in individual instances, can be obtained only by dissection. To diagnose a specific form of lepto-meningitis during life is also very difficult, except, perhaps, in the tuberculous basilary meningitis which occurs in cattle (compare tuberculosis). In this connection we can only add that simple

serous basilary lepto-meningitis is the commonest form of meningitis, whereas the suppurative or fibrinous seems to be much rarer. The diagnosis of a pachy-meningitis, when we have to deal with injuries to the skull, diseases of the ear, etc., appears easier; but even then it can never with certainty be decided in life how far the irritation has extended from the bony covering to the softer membranes and the brain itself. Definite clinical proof of encephalitis can only be established with certainty when being circumscribed, it presents distinctly central symptoms, or when the metastatic development of a cerebral abscess may be presumed. But here, also, we must observe that central or special sense symptoms may be noticed in a number of other brain diseases (hæmorrhage, tumours), and that a suppurative meningitis may metastatically develop instead of a cerebral abscess. These circumstances will explain why we do not here adopt that classification of the different forms of brain-inflammation which we are bound to acknowledge as the best, because most exact, both pathologically and anatomically.

The clinical sub-division of brain-inflammations hitherto found most serviceable was into the acute and sub-acute forms. Under the former were included all rapid cases with extreme phenomena of cerebral inflammation (excitement), and under the second those which presented signs of depression and were also slower in their course. This differentiation is, however, only one of degree and by no means essential. Then, too, every meningitis or encephalitis can, under given circumstances, occur either as acute or sub-acute. Moreover, experience shows that transitions and intermediate stages between the two forms may arise, so that no strict boundary can be established between them. The morbid aspects of acute or sub-acute inflammation of the brain furnish no clue to any uniform process within the brain. The following brain diseases may occur with the aspect of "acute inflammation of the brain," *e.g.*, hyperæmia of the brain, pachy-meningitis, lepto-meningitis (serous, purulent and tuberculous), encephalitis, emboli and, under some circumstances, also tumours in the brain. The morbid aspects of "sub-acute inflammation of the brain" are far more comprehensive, as, besides the cerebral affections just named, other conditions are also included therein, which will be more fully specified in dealing with the sub-acute brain-inflammation of horses. For these reasons we have not been able to decide in favour of this method of sub-division.

I.—INFLAMMATION OF THE BRAIN IN HORSES. LEPTO-MENINGITIS, PACHY-MENINGITIS, ENCEPHALITIS.

General Observations on the Definition of Sub-acute Encephalitis in Horses.—In contrast to the "acute" form in horses which is accompanied by great excitement, a second or "sub-acute," form has been recognised, which manifests itself chiefly by symptoms of brain pressure and stupor. The connection of the two has been indicated by the descriptions: "Inflammation of the brain with mania" on the one hand, and "inflammation of the brain with stupor" on the other. Other synonyms for sub-acute brain-inflammation are: Inflammation of the brain with somnolence, hot head-disease, acute staggers of asthenic character, acute hydrocephalus, semi-acute inflammation of the brain, semi-acute head-disease, passive inflammation of the brain, etc. The multiplicity of these names shows that the "sub-acute" form of the disease is more important than the "acute," and we therefore add a few general observations concerning it.

That it is no uniform disease in horses is plain both from veterinary literature and from the results of dissection. Many practitioners still give this name to all acute or inflammatory brain-affections, the following of which seem rightly to belong to this category:

1. *Lepto-meningitis serosa*, which is probably in its essential a lepto-meningitis basilaris. This form may occur either acutely with symptoms of excitement, or sub-acutely with those of depression. It obviously represents the principal variety both in "acute" and "sub-acute" inflammations of the brain.

2. *Various other brain diseases, both inflammatory and non-inflammatory* in their nature, exhibit also the aspect of a "sub-acute" brain-inflammation, and are therefore probably often included under this title. To these belong *encephalitis, pachy-meningitis, hyperæmia of the brain, hæmorrhage on the brain and tumours of the brain.*

3. *Poisoning by food-stuffs* seems also to have been regarded as inflammation of the brain whenever any specific brain-poison has been generated, perhaps by the attacks of fungi. Here especially lupins, clover (compare lupinosis and clover-sickness (pp. 220 and 222) and leguminous foods must be cited. Also in a few cases the action of narcotics or acrid narcotic plants (equisetum, bearded darnel, yew-tree branches, etc.). The frequent attachment in veterinary literature of blame to leguminous

plants (to clovers, vetches, etc.), as well as the precedent gastric symptoms mentioned therein (colic, loss of appetite, icterus), all point to the connection between "sub-acute" brain-inflammation and poisoning by food-stuffs.

4. Finally, the phenomena sometimes reminds us of an *infectious disease.* The frequent simultaneous affection of several animals, and the suggestion so often made that foul, close stables have been primarily responsible for the outbreak, and above all the absence of any inflammatory changes in the brain or in its membranes on *post mortem*, all produce the impression that we are dealing with a primary, general disorder of the blood, followed secondarily by cerebral symptoms, and therefore resembling an independent brain-affection. In addition to this, dissection proves the existence of other organic diseases altogether characteristic of infectious maladies, such as opaque swelling or fatty degeneration of the liver, decided decomposition of the blood or the formation of ecchymoses. Whether the poison be of a chemical or bacterial nature, or absorbed from without or generated within (auto-infection), is not definitely known. Some authorities assume a peptone-poisoning of the body as causing the "sub-acute" brain-inflammation, and originating in a transformation of the albuminous corpuscles of the blood by the ferment contained in leguminous food. Although this assumption is nothing more than a hypothesis and must be restricted to individual cases, yet it indicates the necessity of a decomposition of the blood in order to account for the disease.

Etiology.—As causes of cerebral inflammation the following factors must be especially noted :—1. The prolonged maintenance of *too high a temperature* in steamy and badly ventilated stables, keeping horses in very warm cattle-byres, abnormally warm weather, sultriness. 2. *Sudden changes in external conditions*, such as change of owner, transport by rail or ship, removal from elevated to low-lying districts, drafting into cavalry, or being placed in a horse show. Cases have been known of horses being affected by brain-inflammation several times in one to two years, and on each occasion when changing owners. Among cavalry remounts and market horses other causes besides the frequent change of surroundings may also contribute, such as over-feeding, over-exertion, chill, bad stabling, etc. 3. *Over-exertion*, especially after long resting in the stable and too high feeding ; the disease is then sometimes observed in the same

stables in which cases of hæmoglobinæmia simultaneously occur. Many horses show symptoms of inflammation of the brain after being kept for a long time on a short head-rope (after embrocation with strong applications and after operations). 4. *Too rich feeding* has always been cited as a cause, and especially with leguminous fodder (peas, beans, vetch, clover), also with food which is too fattening and too nitrogenous, particularly certain kinds of grain, *e.g.*, summer rye. The opinion was formerly held that the occurrence of the disease is exactly proportioned to the leguminous harvest of the district, and that it is unknown where such crops are not grown; an assertion which has been proved untrue. It must, however, be admitted that food which is too rich and nitrogenous produces a predisposition to inflammation of the brain and, in combination with the factors already mentioned, may be classed as one of its causes. 5. *Injury to the roof of the skull,* even simple contusions, also suppurative inflammation of the cranial-bones, especially the temporal bone (suppurative inflammation of the middle ear); serious operations within the cavities of the nose, forehead or upper jaw produce first pachy-meningitis, upon which both lepto-meningitis and encephalitis may supervene. 6. *Parasites, new growths, emboli and thromboses* in the brain may also give rise to brain-inflammation. Among parasites the larvæ of œstrus occur, although rarely, in the brain, and produce general phenomena of brain-irritation or interference with special senses; then also the larvæ of strongylus armatus, and finally, cœnurus-vesicles and cysticerci (compare staggers, p. 660). Of new growths we must mention especially cholesteatomata, which, when very large, may suddenly cause acute inflammation of the brain without any premonitory signs having been observed. The same applies to sarcomata of the ethmoid bone. Emboli are most commonly noticed during glanders and pharyngitis, and usually cause circumscribed brain-abscesses. The formation of thrombosis in the cerebral arteries is also known to provoke an inflammation of the brain. 7. It is also *secondarily* produced by various general disorders and especially by *infectious diseases.* Of these, infectious cerebro-spinal meningitis (Borna sickness) is the most important. Besides strangles and pharyngitis, which may develop by means of emboli into a suppurative encephalitis, septicæmia and pyæmia are particularly liable to be complicated with suppurative lepto-meningitis; thus, for instance, in the "joint ill of foals." Brain-inflammation is also noticed in the course of pleuro-

pneumonia and variolous fever. Whether bacteria play a part in the etiology of cerebral inflammations, and if so, to what extent, is undetermined.

Horses not yet fully grown show a predisposition to the disease, for which reason its occurrence is most frequent between their second and sixth years. But even very old and quite young animals may also be attacked. Horses, moreover, descended from sires or dams which have previously suffered from inflammation of the brain or staggers show a decided liability to the complaint. Definite data respecting the influence of race are not yet to hand, but languid, common country horses seem to be more readily affected than those of higher breed, though the complaint is fairly common among them also. Military horses, though not exempt, as some have affirmed, are but seldom attacked, a fact probably due to their regular feed and keep. Though the malady may occur at all seasons of the year, it is more prevalent in the spring and early summer. In some districts it appears to be almost enzoötic, and in certain stables even permanent.

Pathological Anatomy.—1. **Pachy-meningitis** when *acute* is usually suppurative. The dura mater is found to be hyperæmic, infiltrated with hæmorrhages, its inner surface opaque and covered with fibrinous or purulent deposits ; sometimes the dura has a fleshy appearance. *Chronic pachy-meningitis* is characterised by fibrous proliferations of connective tissue, which result in adhesion of the dura to the roof of the skull ; at the same time, bony new-growth takes place (P. ossificans).

2. **Lepto-meningitis** (*serous*) begins with hyperæmia of the vessels of the pia, and swelling, opacity and a spotted or striped hæmorrhagic reddening of the soft cerebral membrane. Between the pia and arachnoid or dura we find an accumulation of yellowish-red fluid (hydrocephalus externus), which, if abundantly present, flattens the cerebral convolutions. The brain-substance directly beneath the pia is œdematous, its sectional surface showing a moist lustre, and, if it be pressed, a little serum gathers later in the hollow thus made. This œdematous saturation may develop into complete softening, when the brain-matter takes on a pulpy consistence ; occasionally hæmorrhage is found in the brain. In the ventricles serum has accumulated, usually bright and clear, but sometimes dull and bloody, and varying much in quantity (the average is 1 to 1½ oz.,

but sometimes only a few drams). According to Schütz, out of twenty-five cases of acute hydrocephalus he found in the lateral ventricles from ⅛ oz. to 1⅛ oz. in twenty-two instances, once 1⅜ oz., once 2 oz., and in one case 2⅜ oz. of the fluid. When there is much of this, the optic thalamus and the corpus striatum are flattened and the ventricles dilated; the cerebral substance around the ventricles is œdematous and softened. The network of veins is also œdematous, swollen, and forms flabby, greyish-yellow and puffy lumps.

Suppurative lepto-meningitis is usually a convexity-meningitis. In addition to hyperæmia we find hæmorrhagic redness and swelling of the pia between this latter and the arachnoid; also between arachnoid and dura greyish-yellow, flaky or moderately firm exudation, which consists mainly of pus-corpuscles and finely granular fibrin, and is also croupous in its nature. The next adjoining portion of the cerebral cortex is sometimes hyperæmic, sometimes anæmic and œdematous, or even purulently infiltrated (meningo-encephalitis). The ventricles of the brain are usually empty.

Chronic lepto-meningitis is manifested by adhesion of the arachnoid to the dura, by milky opacity and thickening as well as internal adhesion of the pia to the brain-substance, into which it projects strong processes of connective tissue. The veins are dilated and their walls thickened; sometimes both chronic œdema of the pia mater and serous effusion and atrophy of the upper surface of the brain are visible. Also in the ventricles we may occasionally find small quantities of fluid, considerably dilated vessels of the ependyma with thickening of the membrane itself.

3. **Encephalitis** is generally partial or circumscribed; rarely is it general or diffuse.

(*a*) The *circumscribed non-suppurative* form presents irregular round foci, which are usually from the size of a pea to a hen's egg, but may also embrace an entire cerebral lobe and are not sharply defined from their surroundings. At first the places in question are almost imperceptibly diffusely or distinctly spotted with red, owing to hyperæmia or hæmorrhage. To this are soon added, as a result of serous exudation, swelling and softening of the brain-substance, when the microscope shows the cells of the neurolia (neuroglia), as well as the ganglionic cells to be swollen, granular opaque and in a condition of fatty degeneration, the axis-cylinders irregularly changed and the tissue of the glia

infiltrated with small cells. The encephalitic focus undergoes maceration, swelling and breaking-down, and finally represents a softened substance consisting of decomposed and fatty-degenerated glia and ganglion-cells, white blood-corpuscles and free nodules of fatty granules, and is then described as a simple brain-inflammation or inflammatory softening of the brain. It may be distinguished from the non-inflammatory form, caused by disturbed nutrition, by the infiltration of white blood-corpuscles, which is its characteristic sign. At this stage the inflammatory process may terminate. It is often complicated with hæmorrhage, as the vessels traversing the encephalitic focus may undergo fatty-degeneration and rupture, which condition is known as hæmorrhagic encephalitis, or inflammatory red softening of the brain. As the colouring matter of the blood becomes decomposed in the above-named focus, it gradually loses colour and appears yellowish—this is inflammatory yellow softening of the brain. The decomposed and exuded matter may liquefy and be re-absorbed, forming then either a grey, gelatinous focus of disintegration (grey inflammatory softening) or a cyst, in some cases, a sclerosis of connective tissue, or a cicatrix.

(*b*) *Partial suppurative encephalitis* appears as an abscess in the brain. It develops especially after septic or pyæmic emboli from hæmorrhagic inflammation centres, which gradually enlarge and undergo purulent disintegration. A granulating membrane may form around the abscess, which shuts in the pus-cavity. This pus is at times yellowish, greenish or discoloured; its consistence is that of cream. Such abscesses of the brain are most frequently observed in strangles of horses, and also in cattle. Usually there is one abscess only, but occasionally several are found, and in very rare cases a large number in the same animal.

(*c*) *Diffuse encephalitis* is very rare. Friedberger observed in one horse, which during life showed the customary signs of inflammation of the brain, no changes in the brain beyond a diaphanous, yellowish colour like amber spread over the white matter of both frontal lobes of the large cerebral hemispheres. The microscope showed that the peri-vascular cavities as well as the neuroglia were closely permeated by white blood-corpuscles; the capillaries appeared hyperæmic. No other changes could be perceived.

The accumulation of Serous Exudation in the Ventricles in Lepto-meningitis.—The quantity of fluid varies considerably. Even in sound horses, according to Hering, the quantity of cerebral and cerebro-spinal fluid fluctuates to a remarkable extent, and in sick horses this is still more marked. Thus, in four horses with brain disease, the total amount of cerebral and cerebro-spinal fluid was $4\frac{1}{8}$–$9\frac{1}{2}$ oz. (the cerebral fluid alone weighing $1\frac{1}{8}$–$4\frac{1}{8}$ oz.), while in the case of three other sick horses the quantity of fluid present amounted to $10\frac{1}{2}$–$14\frac{3}{8}$ oz. Hering rightly concludes from these facts that the symptoms in brain disease are not caused by what is known as "brain-pressure." Johne found an average of $5\frac{1}{2}$ oz. of fluid in sound horses, and in horses which had died of diseases other than brain disease, he found an average of 7 oz. According to Johne, if the amount of cerebral-spinal fluid in horses with brain disease is to exercise any deleterious effect by actual mechanical pressure, it must be at least one-third larger than the figures given above. He also points out that the anatomical changes which are said to be characteristic of sub-acute inflammation of the brain (serum in the ventricles and sub-dural spaces; punctate hæmorrhages, etc.) are very often present in horses which have died from other causes (symptoms of engorgement or due to cardiac weakness). Also a very common mistake is made with regard to the communication between the ventricles and the sub-arachnoid spaces. As a matter of fact there is no direct communication between them (Menault, Perosino, Hering, Gurlt, Franck, Seisering, Müller). It is also impossible for the cerebro-spinal fluid to escape from the ventricles after decapitation. The amount of fluid found in the ventricles at the autopsy represents with absolute accuracy the condition during life. When the head is severed, the fluid escapes from the sub-arachnoid spaces and this is often mistaken for exudation from the ventricles.

Symptoms of Inflammation of the Brain in Horses.—The phenomena of this malady in horses are exceedingly variable, sometimes the signs of excitement predominating and sometimes those of depression, while the two are frequently commingled. Both the degree, form and seat of the inflammation cause great variations, as do also the temperament and intelligence of the affected animal. For these reasons no typical picture of inflammation of the brain can be drawn, and we must restrict ourselves to a delineation of the several symptoms.

1. *Signs of excitement* generally appear in the severer cases, but they may be absent, or not visible until later. They consist in restlessness and excitement, which usually come on unexpectedly and suddenly in the stall, or when being ridden or driven, and may amount to raging mania. The horse presses or leaps forward or sideways, rears, hangs upon the halter or snaps it, tumbles down, rolls over or strikes its head against the wall, by which means it may receive serious injury both of head, breast and limbs. It can no longer be led, or only with

difficulty, has a hasty, thrusting, groping movement, runs at every obstacle and, if once taken out of the stable, can only with much trouble be got back again. The eye is wild or staring and its pupil contracted. The papilla of the optic nerve is found, on examination by the ophthalmoscope, to be redder and its vessels more highly charged. Sensibility is heightened; a touch upon the head or ears, a word spoken, or even the slightest sound, increases the attacks. Muscular spasms are also observed, especially in the facial muscles, as twitching and tremor of the lips, or as spasms of the jaw (grinding the teeth); also in the muscles of the neck and extremities and at times also of the diaphragm (sobbing). Stallions often project their penis and show erection.

2. *Symptoms of depression* occur either in connection with those of excitement, or at the commencement of the outbreak; but in extreme cases which rapidly end in death they may be absent. Sometimes they appear suddenly, at others after certain premonitions, such as lassitude, laziness or loss of appetite, and reveal themselves chiefly in a sleepy, unconscious condition. The head is hung down or rested on the manger, the animal leans against the stall, its eyes closed as in sleep, with the pupils dilated, and will remain thus for hours, or for half a day; or it may adopt unnatural postures, crossing its legs, or drawing them all into a bunch, or perhaps straddling them wide apart. Sometimes it loses its balance and falls over, or doubles up its joints, or stumbles forward, especially when trying to pick up food. It cannot be roused from this sleepy state, or only for a short time and by dint of much shouting. Rarely does the horse lie down, and then can only with difficulty be made to rise again, when it sometimes remains a long time on its knees. Its gait is uncertain, stumbling and tottering, the legs being either lifted too high or not high enough; and rotary movements are often observed, lasting even for hours and mostly in one direction. Sensitiveness may also be much diminished, and one may tread on the coronet joint, pull the ears, clap the hands before the animal's face or strike it with the whip without eliciting any sign. Finally, symptoms of paralysis may develop, *e.g.*, paralysis of the retina or centre of vision (amaurosis) of the ear, pharynx, upper eyelid (ptosis), hind-quarters; and also, in encephalitis, of the muscles of one side or half the side.

3. *Fever* is usually present and may rise to 104–106° Fahr., shivering fits may also be observed, especially in suppurative

locally and generally. To the immediate neighbourhood of the brain apply ice, snow or cold-water bandages, or, if necessary, a so-called cooling mixture of saltpetre, common salt and glauber salt with vinegar or water, and continue these as long as the signs of abnormal flow of blood to the brain are maintained. Later on, when symptoms of stupor predominate, pouring cold water on the head is better ; this should be done with a watering-can and from different heights, as this has also a stimulating effect. These cold bandages should be helped by cold infusions of the rectum, whenever this can be done. The practice of blood-letting formerly adopted in all cases of brain-inflammation is permissible only at the outset of the disease, or when pronounced symptoms of active hyperæmia of the brain are present. In all other cases it must be avoided, because, if exudation has already taken place in the brain, it only increases the anæmia of the brain caused by pressure of this exudation, and thus exacerbates the disease.

Derivation to the bowels is also prescribed. Here, as in hyperæmia of the brain, we can recommend only the true drastics (croton oil, aloes, tartar emetic) for the beginning of the malady, and only when real hyperæmia to the brain is still present, and the animal's constitution permits their use. In other instances drastic remedies, according to our experience, are just as injurious as bleeding, as by diverting the blood too much to the intestines they only increase the anæmia and depression of the brain. On the other hand, gentler purgatives (neutral salts, or smaller doses of calomel) are often useful, especially if the bowels be obstructed, and even if depression has already set in ; that is, if it be found possible to introduce them by the mouth. If this cannot be done, then recourse must be had to subcutaneous injections of physostigmine (1½ grains per dose), or arecoline (1½ grains).

Attempts are also made to cause resorption of the fluid which has collected in the ventricles and between the membranes of the brain, by administration of pilocarpine, which is usually injected for several successive days in doses of from three to eight grains. Although pilocarpine renders good service in many cases of acute and sub-acute hydrocephalus, yet experience has shown that it is not an infallible remedy, but often leaves the practitioner in doubt, or may even aggravate the case. We advise caution against using it in too large doses. The same warning applies to arecoline (1½ grains).

Derivation to the skin by sharp inunctions of croton oil,

mustard oil or cantharides-ointment near the neck or on both sides of the throat are objectionable during the stage of excitement, as they tend to increase it. Their use in conditions of stupor is also doubtful, so far as our own experience goes; the same applies also to the cautery and setons. Other authorities, on the contrary, highly recommend inunctions and derivations. For conditions of extreme excitement the administration of bromide of potassium or chloral hydrate 6 to 12 drams (rectal) is to be recommended. The sick animals must not have rich or too stimulative food; as a rule we give them no oats, but bran, meal-drinks, green fodder, turnips, etc. Prophylaxis consists in avoidance of the causes already mentioned; for full-blooded and well-nourished horses the occasional administration of a purgative is advisable, or a daily dose (6 to 12 drams) of artificial Carlsbad salt. Straw hats should be used to protect the head from the sun.

Dexler believes that acute lepto-meningitis in the horse is usually of infective origin, the agent which causes the disease being conveyed to the brain from some other affected portion of the body (pyæmia). Occasionally the source is unknown. The effects of temperature, change of food, over-strain, etc., are contributing causes at the most (?). According to Dexler, the anatomical variations are solely microscopic (small-celled infiltration). He also considers that encephalitis is nearly always infective in its origin (contagious pleuro-pneumonia, glanders, pyæmia, septicæmia, unknown contagia). The principal macroscopic change—namely, red softening of the substance of the brain—may be absent, and in such cases diagnosis can be assured only by the aid of the microscope. A peculiarly acute form of encephalitis (sub-pial and sub-ependymal hæmorrhages) with fever, bears, according to Dexler, an etiological relationship to acute hydrocephalus and even staggers. Dexler also describes these forms of brain disease as contagious.

II.—INFLAMMATION OF THE BRAIN IN OTHER DOMESTIC ANIMALS.

This disease offers in other animals the same general conditions in regard to etiology, pathological anatomy and therapeutics as in horses, yet the malady varies somewhat in different animals, so that its symptoms must be briefly discussed.

1. In **cattle** the excitement manifests itself in bellowing, snorting, shaking the head, thrusting with the horns, which may even be broken off, gaping, foaming, leaping into the manger, charging and overthrowing objects lying in the path,

trembling, epileptiform spasms, crib-biting and gnawing, a glaring look in the eyes, rolling the eyes, spasmodic uplifting of the legs, rotary movements, butting forwards and striking the head against the wall. Later on we find stupor, a groping gait, unphysiological attitudes, lessened sensibility, collapse, general signs of paralysis and amaurosis. The course is generally very acute, and the condition generally ends in death within a few hours or days. Confusion may occur especially with malignant catarrhal fever (head-sickness of cattle); but differentiation is generally possible by noting the affections of the eyes and of the nasal and frontal cavities. Acute lead-poisoning and poisoning by corn-poppies may also present similar symptoms.

2. In **dogs** brain-inflammation generally exhibits premonitory signs, which are restlessness, timidity and a desire to hide. The dogs no longer know their masters, sometimes howl continuously, or scream if touched. They seldom bite, and only when seized; indeed, aggressive conduct towards man or other animals has never been observed. Fits of mania are very rare, and seem only to arise when the dog is fastened in its cage, when it will bite at the bars and tear up its bed. In the open it runs aimlessly about and knocks against things so violently as to be thrown down. Its glance is wandering, the conjunctivæ are very red, the skull feels very hot, the pupils are contracted; vomiting also and staggering, epileptiform spasms and even involuntary movements are noticed. Later the dog lies paralysed, stupefied and unconscious. The result is often quickly fatal; but chronic conditions of depression may also develop, viz., hydrocephalus, amaurosis, deafness, etc. Inflammation of the brain is much rarer among dogs than horses, the statistics of the Berlin dog-hospital showing only 103 cases out of 70,000 dogs treated.

Confusion may arise especially with rabies; and often autopsy only can decide the question. In brain-inflammation we lack the aggressive action, the altered voice and the consumption of indigestible substances; while rabies does not show such intense injection of the conjunctivæ, or so high a temperature of the skull. A similar aspect is also presented by nervous distemper, pentastoma tænioides, worms in the bowels and several forms of poisoning (filix and santonin).

3. **Pigs** show attacks of mania, climb up the walls of their

sties, utter piercing cries, grind their teeth, dribble, fall into convulsions, have fits of twitching and epileptiform spasms, twist round and round, press hard against the side of the sty or other object, sometimes collapse entirely; their skulls and the root of their ears feeling very hot. Later they hang down their heads and show signs of stupor and paralysis. Pregnant sows abort. The end is frequently fatal, recovery being rare; a condition of sleepy staggers may, however, remain behind.

4. **Sheep** are said to hold their heads stiffly in one position, press forward, twist about, stagger and show twitchings. The complaint is not easy to distinguish from staggers (cœnurus cerebralis); in general, though, the course of the latter is more chronic.

5. **Goats** also hold the head and neck stiffly, grind their teeth and have epileptiform spasms. In sheep and goats it is not always easy to decide whether the symptoms arise from simple inflammation of the brain or from cerebro-spinal meningitis.

SLEEPY STAGGERS (*Chronic Hydrocephalus*).

Occurrence.—Chronic dropsy of the cerebral ventricles is most common in horses, in which it represents the chief cause of sleepy staggers; and is rarer in cattle, dogs, pigs and sheep. Aged horses are most subject to it, as it rarely appears earlier than their fourth year; geldings, moreover, seem to be more prone to attack than stallions or mares. This is perhaps due to the deterrent effect of castration upon brain development, which has been scientifically demonstrated in several animals; and any organ which has been checked in development has naturally less power to resist external influences. It is also universally believed that common breeds, such as those from low-lying, often-flooded regions, with weak constitution, dull and phlegmatic temperament, broad heads yet narrow skulls, have a special disposition to chronic dropsy of the ventricles.

Etiology.—It is customary to distinguish between two forms of chronic staggers (hydrocephalus) in the horse, namely, primary and secondary staggers.

1. Secondary staggers is the result of a previous cerebral

inflammation accompanied by acute or sub-acute hydrocephalus. As soon as hydrocephalus becomes chronic, it passes into the condition known as staggers (so-called secondary, deuteropathic, or symptomatic staggers).

2. The causes of so-called primary or idiopathic staggers, *i.e.*, of a gradually and slowly developed staggers independent of the above-named factor, are not yet clearly known. Dexler advances the theory that an idiopathic or primary hydrocephalus does not occur in the horse. He maintains that the condition always appears in its secondary, mechanical form, being brought about by the passive engorgement of the cerebro-spinal fluid in the ventricles of the fore-part of the brain, as a result of the occlusion of the aqueducts of Silvius. By many the disease is regarded as congenital (Gerlach, Haubner). In France also, it is said that sleepy staggers was almost enzoötic in certain stables of the Alps and Rhone valley as a result of heredity, so that breeding had to be entirely relinquished. By others, again, it is denied that heredity is a factor in the etiology of staggers (Dieckerhoff, Dexler).

As accidental contributory causes repeated active and passive cerebral hyperæmia, with great heat, over-exertion and over-feeding, particularly with cereals and leguminous food, have always been blamed, especially if there be also plethora, liver or other gastric affections, disorders of the heart and lungs which disturb circulation, compression of the jugular vein by the harness, or the use of too severe a bit.

Post-mortem Conditions.—The anatomical changes in chronic dropsy of the brain-ventricles consist first in the collection of a serous fluid, as clear as water, in the lateral ventricles of the brain, and sometimes also in the third ventricle. These lateral ventricles are thereby more or less enlarged, sometimes to a considerable extent, so that perforation of the dividing septum may occur, allowing free passage from one to the other. The optic thalamus and corpus striatum, as well as the cornu ammonis, become flattened by the pressure of this liquid (atrophy of the brain), and the olfactory bulbs are sometimes bulged out into thin sacs. The ependyma of the ventricle is thickened and made rough and uneven by deposits of connective-tissue granulations; the adjoining brain-substance appears anæmic, pale in colour, dryer and harder. The venous plexus is thicker, gelatinous, swollen and shows

dilated and sinuous vessels. The volume of the brain appears to be increased, its convolutions are flattened, the furrows smoothed out and the superficial cortex is dry and anæmic. If the cerebral mass be removed to the roof of the ventricle, the latter bulges upwards and, upon puncture, the contents squirt out in a stream. The occurrence of the morbid symptoms is easily explained by these anatomical changes; on the one hand the pressure is exerted by this fluid matter, and on the other the partial decay of the brain-substance.

Dexler describes the anatomical changes in acquired hydrocephalus internus in the horse as follows:

"In the greater number of cases there is moderate symmetrical dilatation of the central cavities of the thalamencephalon and of the occiput. This dilatation involves the anterior half of the third ventricle, together with the recessus opticus and the pineal gland and including the central portion of the lateral ventricles. The enlargement of the inferior cornu depends upon the presence, which is by no means rare in the horse, of agglutinations of the ependyma in that region. But even in such cases a considerable dilatation is occasionally found which may be accompanied by a partial curling-up of the edges of the coverings of the hemispheres. The increased circumference of the cerebrum is shown by the flattening out of the frontal convolutions and of the tentorial surface of the occipital lobes; in the formation of a swelling due to pressure, with consequent depression of the corpora quadrigemina; complete obliteration of the aqueduct of silvius; and diminution in the size of the recessi infrapinealis and infrafundibuli. The mesial section of the thalamencephalon is reduced; the transverse section of the massa intermedia is elliptical and the posterior lower quadrant of the third ventricle is much contracted. The entire mid-brain, together with the pons, is shifted slightly back into the cerebellar space, and pressed against the base of the skull, so that the exterior surfaces of the crura cerebri appear quite smooth. The sulcus subst. perf. post becomes narrower and deeper, and the trunks of the third, sixth and seventh pairs of cranial nerves are flattened.

"In very severe cases, all these abnormalities become to a certain degree intensified. The lymph spaces get filled up, the base of the thalamencephalon is so distended that the pituitary body appears to lie in a deep cleft. The mamillary body may disappear partially or entirely, while the uncus becomes more readily visible than in the normal subject. The chiasma is smoothed out upon its ventral side and the optic thalami become distended like bladders. The displacement of the middle brain is more apparent and is accompanied by a partial curling up of the anterior edge of the slightly flattened pons. The enlargement may be so great as to press down the superior vermiform process and displace the entire cerebellum towards the foramen magnum.

"In long-standing cases, the enlargement of the ventricles may be very pronounced. The septum pellucidum may be thinned to an extraordinary degree, or it may even be perforated, while the central ganglions and the middle brain may be so squeezed together as to be scarcely

recognisable. The distension may be so great as to reduce the cerebellar space and compress the organs contained in it. The paramedian fissures are smoothed out, the superior vermiform process is pressed down, while the inferior vermiform process is reduced to a blunt ridge, which reaches to the foramen magnum over the medulla and almost entirely closes the subarachnoid spaces. The ventriculi olfactorii are extended to their greatest normal width, but their convolutions are unchanged. The bony structure of the skull may in places become slightly thinner. Slight obstruction of the great veins is occasionally present, though this is not the rule. The plexus and the ependyma are stretched, but show no specific abnormality. The cerebro-spinal fluid is always clear and does not appear to be increased; exact quantities are unknown. The amount of fluid in the dilated ventricles is very little more than under normal conditions. It is rare to find signs of abnormal pressure in the ventricles after death.

"Between the two forms of the disease described above, which owe their dissimilarity to the difference in the duration of the morbid process, there are numerous transition stages. But however much the anatomical appearances may be varied by the absence or suppression of one or other of the less constant abnormalities, the *post-mortem* findings invariably include three principal changes—dilatation of the ventricles of the fore-brain, deformity of the middle-brain, and swelling due to pressure."

Symptoms.—The aspect presented by chronic hydrocephalus or sleepy staggers is chiefly a disturbance of consciousness, perception, volition and sensation, with unusual actions and movements on the part of the animal; also secondary disturbance of the heart's action, of digestion and respiration—other conditions being normal and especially the absence of fever. But the malady may occur in varying degrees and with all sorts of complication of the several symptoms. The most important signs in horses are:

1. *Disturbance of Consciousness*, or sense of individual existence. This is revealed by the eye and bodily attitudes. The former is often half shut, the upper eyelid being drooped, and the glance is frequently quite expressionless, fixed and dull; the animal looks as though sleepy. The head is generally sunk, or else laid upon the bars, manger or knees, or perhaps leans against the wall. The position of the legs is exceedingly strange and unnatural; sometimes they are withdrawn far under the body or even all to one point, at others, they are crossed, or are planted in front, or one upon the other. They can, moreover, be placed in these or other positions at will, and will often remain thus for a very long time. These disturbances of consciousness increase if the animal be moved. Horses afflicted with sleepy staggers frequently manifest a strange desire to thrust either to the right or left, and this

dulness of the sensorium is always greatest immediately after exercise.

2. *Disturbances of Perception, i.e.,* of its relation to the outer world. The animal takes no notice of its surroundings, or gains false impressions of them, which facts are first shown by the erratic motion of its ears. This is variable and contradictory, being not towards a sound but away from it, and sometimes one ear listens backwards and the other forwards. This disturbed perception usually betrays itself in the taking of food and drink. The fodder is first seized, perhaps, in large bunches, chewed for a while, and then allowed to rest quietly in the mouth, as though chewing were forgotten; then it will be taken more slowly and fully masticated, though with frequent pauses. Generally, also, the horse feeds rather from the ground than from the rack, which may perhaps arise from the altered pressure of the fluid when the head is raised or sunk. Many horses when suffering from sleepy staggers stand too near the manger; they pull hay from the rack by jerking the head, while twisting it strangely to one side. In drinking, the head is plunged much too deeply into the bucket, often over the nostrils, and the water is actually chewed. When walking, the legs are frequently lifted very high, as though some obstacle were to be stepped over, and regular circular movements may be observed. In being backed, the horse drags its fore legs after it instead of raising them.

3. *Disturbed Volition.* This is shown in various ways. The sick horse obeys an order to move very slowly to the right or left in his stall. Sometimes he will not move, is very dull and lazy at his work, must constantly be urged on and can only with great difficulty be got to move backwards. He will often refuse to do so, will rear when urged, and, while obeying neither rein nor whip, attempt to constantly move to one side.

4. *Disturbances of Sensation* are chiefly shown in diminution and dulness of the same. One may step upon the animal's coronet without it taking any notice, or may pull its ears, pinch its flanks, or clap the hands before its forehead, with hardly any result.

5. *Secondary Disturbances of the Heart, of Digestion and Respiration.* The pulse is soft, slow and often below its normal number, peristalsis is frequently interfered with and suppressed, as is also evacuation; breathing is deep and slow and its frequency much below the normal rate.

Course.—The course of sleepy staggers is chronic, the condition often lasting for years and even until the animal's death. In other cases, distinct improvement or retrogression has been observed. The latter occurs mostly when unfavourable circumstances have reacted on the animal (over-exertion, too rich food, steamy stables, great heat). Thus in summer such horses often seem much worse, as compared with other seasons. Favourable influences, such as cool, airy stables, suitable diet and careful handling, may cause marked diminution in the symptoms. Such a temporary improvement frequently sets in after the use of pilocarpine and arecoline. The symptoms may, indeed, almost disappear, and this has often been mistaken for a sign of ultimate recovery.

Sometimes chronic hydrocephalus is complicated with congestion and even inflammation of the brain, especially when the chronic has been developed from an acute, inflammatory form. We then obtain the aspect of staggers plus that of brain-congestion or inflammation—which latter predominates. These fits of excitement, mania and raving are sometimes evanescent (congestion of the brain), occasionally more prolonged (inflammation of the brain). Formerly this complication was called "raging" or "erethistic" staggers, a name which should no longer be retained, and which means nothing but an acute (hyperæmic or inflammatory) process upon a basis of chronic hydrocephalus. Further description is unnecessary, as it corresponds entirely with hyperæmia and inflammation of the brain. Though chronic hydrocephalus is not itself fatal, yet when complicated with cerebral inflammation, it may frequently have such a termination.

Chronic Dropsy of the Ventricles in Other Animals.—The symptoms are not very different from those of sleepy staggers in horses. Cattle, pigs, dogs and sheep show persistent stupidity, heaviness in the head, and seem almost imbecile; thrust to one side, grope their way uncertainly, run against objects (amaurosis), are deaf and deficient in power of smell; digestion also is disturbed, but there is no rise of temperature. The sickness is generally called "stupidity," and in pigs sometimes "silliness." It is not unfrequently a sequel of distemper in dogs, and is occasionally congenital. The Berlin dog hospital reported 29 cases out of 70,000 dogs treated in nine years.

Therapeutics.—No treatment is of any avail. The remedies formerly used and praised, including perforation of the olfactory bulbs in order to empty the brain-ventricles (Heyne), have all been proved useless. Pilocarpine and arecoline are

also of no avail when the condition (hydrocephalus) is once fully developed, as there are no means of compensating the atrophy of the brain caused by pressure. The utmost they can do is to produce a temporary psychic improvement and thus render the animal a little more serviceable. By prudent prophylactic measures we may prevent the disease from growing worse, and especially ward off its dangerous complications with hyperæmia and inflammation of the brain. With cool and airy quarters, we must particularly see that the animals are not over-worked and not too richly fed. They should have laxative foods (bran, green food, turnips) and even gentle laxative medicines, the latter especially in summer ; for instance, a little neutral salt occasionally.

Sleepy Staggers as a Warranty-Defect.—Sleepy staggers is more important in its character as a defect against which warranty can be demanded than in its clinical aspect. Although this forensic question scarcely belongs to a work on the medicinal treatment of animals, yet the chief points observed in judging sleepy staggers must here be summarised. The disease is forensically defined as a non-febrile, chronic, incurable brain-disease of horses, which manifests itself in disturbance of consciousness, of perception, of volition and of sensation, and may vary greatly in its degree and complications. Its cause is usually chronic dropsy of the ventricles of the brain. In rare cases the following pathological processes in the brain can also produce the morbid aspect of sleepy staggers : hydrocephalus chronicus externus, pachy-meningitis and lepto-meningitis chronica, adhesion of the cerebral membranes to each other, to the brain or to the roof of the skull, exostosis of the cranium, chronic encephalitis with softening or sclerosis, ependymitis sclerosa, new growths in the brain or venous plexus, *e.g.*, cholesteatomata (but which often show no symptoms), psammomata, melanomata, œdema and parasites in the brain (hydatids). Diagnosis is easy when the symptoms are pronounced, not otherwise ; but some of these difficult cases may be judged according to their subjective conception. The animals (having no fever) are examined first at rest, then during and after exercise (riding, driving). One has to consider, not only solitary phenomena, but all in their relation to one another, and special weight must be laid upon diminution of usefulness. This always exists in horses subject to sleepy staggers, even though for some kinds of work they may be used for a long time ; for general traffic they are always dangerous, because unreliable and subject to inflammation of the brain. By way of differential diagnosis the following diseases must be distinguished from sleepy staggers :—1. Congestion and inflammation of the brain, which are characterised by injection of the mucous membranes of the head, high temperature of the skull and symptoms of excitement. In particular, acute hydrocephalus is distinguished by its acute and often feverish course, its combination of depression and exaltation, of anæsthesia and hyperæsthesia, by loss of appetite, variability of the pulse, and distinct post-mortem lesions (hyperæmia and inflamma-

tion). 2. Severe and general fevers, especially infectious diseases (influenza, murrain, glanders). 3. Affections of the brain accompanying the change of teeth (gums reddened, mucous membranes of the head injected, youth) and change of coat. 4. Sexual excitement, so-called "seminal" and "maternal staggers" (periodical and connected with the sexual life). 5. Gastro-intestinal catarrh, liver-complaints, etc., so-called "stomach" and "liver-staggers." (Compare Schweinsberger Sickness, p. 277.) 6. Phlegma, weariness, senile weakness, deafness, blindness.

According to German law, the period of warranty is fourteen days.

HYPERÆMIA AND ANÆMIA OF THE BRAIN.

Etiology.—Hyperæmia of the brain is conveniently divided into congestive or active hyperæmia, with a rapidly occurring fluxional excess of blood in the brain; and an engorged or passive hyperæmia, with accumulation of blood in the brain owing to defective or impeded outflow of the blood.

1. **Congestive or Active Hyperæmia of the Brain** occurs especially in young animals, and most frequently in horses, dogs and sheep, many animals seeming to possess an individual predisposition. Its causes are: severe bodily exertion, driving or riding too fast, over-strain during breaking-in; extreme psychic excitement, *e.g.*, during the sexual period, a railway journey (so-called railway sickness), or after a change of quarters; abnormal activity of heart following hypertrophy; shock or injury to the brain and its membranes; prolonged action of strong sunlight upon the head (sun-stroke insolation), excessive summer-heat, hot, stuffy stables, severe chills, sudden changes of weather; the process of dentition, especially in dogs and horses (but to which, as a rule, too much importance is attached); collateral congestion of the carotid arteries, as a result of the exclusion of important arteries of the general circulation, as in thrombosis of the arteries of the thigh and pelvis (intermittent lameness), or in compression of larger arterial capillary areas by abnormal gas-pressure in the stomach and bowels (acute flatulence of ruminants, wind-colic of horses); excessive eating after previous fasting or with but little work, indigestible food (leguminous, summer-wheat, rye, clover).

As a secondary symptom congestion of the brain occurs in the course of many diseases, such as in brain and infectious diseases (oftenest, perhaps, in the distemper of dogs) and in

poisoning by narcotic and narcotico-acrid substances. Finally, previous brain-affections (inflammation, chronic dropsy of the ventricles) seem to leave behind a predisposition to hyperæmia of the brain.

2. The causes of **engorged or passive hyperæmia** consist in local compression of the jugular veins by tight collars, throat and head-straps, by securing the head too firmly when breaking-in, by tight bearing-reins, or by a very large struma (goitre), especially in dogs. Moreover, non-compensated valvular defects of the heart, as well as destructive changes in the lung (emphysema, induration) and compression of the lungs by accumulated fluids in the pleural sac all tend to impede the outflow of blood from the veins of the brain. In cases of abnormal gas-accumulations in the stomach and bowels a passive hyperæmia of the brain is set up along with the congestive form.

3. The causes of **anæmia of the brain** (by which we must always understand an arterial anæmia) are weakness of the heart; compression of the carotids by tumours or after ligation; embolic or thrombotic stoppage of the brain-vessels; diminution of the total amount of blood in the body, or of its most important constituents (anæmia, severe hæmorrhage, chlorosis, leucæmia); a too rapid flow of blood from the brain to other parts, *e.g.*, upon suddenly emptying a pleuritic exudation or gas-accumulation from the stomach or bowels; an increase of inter-cranial pressure caused by compression of the brain by accumulations of fluid, tumours or effusion of blood. A reflex spasm of the vessels of the brain after the action of external irritants can also, perhaps, as in shock, be regarded as a cause of anæmia of the brain. Finally, it forms an accompaniment of many diseases associated with poverty of blood. As passive hyperæmia of the brain always brings with it an arterial anæmia, it may also to some extent be regarded as a cause of the latter.

Post-mortem Conditions.—1. *Hyperæmia of the brain* is marked by great fulness of the brain-vessels and of its membranes, with which small hæmorrhages may also be present in the soft cerebral substance itself. The grey matter of the brain is a bluish-grey, and the white is dull grey to greyish-yellow. The cut surface of the brain shows a large number of blood-dots, which may be ranged in rows and, in contrast to the

hæmorrhagic foci in apoplexy of the brain, can be easily wiped off. The entire brain appears larger, in consequence of which, when the hard cortex is incised, it bulges somewhat. In severer stages of brain-hyperæmia, transudation of serum into the peri-vascular lymph-cavities takes place, and even into the cerebral tissue itself, whereby the capillaries of the brain are compromised. Viewed as a whole, this condition of cerebral œdema is characterised by poverty of blood in the entire organ, a singularly watery lustre upon the cut surfaces, and even the oozing therefrom of serum, and also by the diminished consistence of the brain substance. After frequently repeated hyperæmia, opacities and thickenings of the soft substance often remain, with enlargement of the vessels and thickening of the ependyma. Possibly the so-called Pacchionian granulations upon the arachnoidea are produced by chronic hyperæmia of the brain.

2. *Anæmia of the brain* consists in a notable emptiness of the blood-vessels of the brain and of its membranes, as a result of which its grey matter appears remarkably clear and colourless, and its incised surface shows only very few blood-dots. In cases of chronic anæmia the substance of the brain itself has often a somewhat tough consistence.

Symptoms.—1. In the **active form of hyperæmia** of the brain these are shown in paroxysmal appearances of excitement, in which disturbances of the psychic, of the sensory or motor spheres predominate, usually to be soon followed by signs of depression. The several aspects vary greatly according to the intensity and locality of the hyperæmia, or the individuality and species of the animal. The excitement stage often commences suddenly, but in some cases gradually, with signs of restlessness and agitation, which may grow to fury and mania. *Horses* plunge forward against barriers and walls, striking them with their heads or breasts and frequently sustaining serious injury; or they rear, strike out with the fore feet on to the rack-chain or manger, kick out without cause, show their teeth, bite neighbouring objects, neigh and shake their heads. Others run back suddenly in the stall, snap their chains and then generally fall backwards, or hang themselves in the halter. They are at the same time very timid, and extremely sensitive to external influences (touch, noise, light); sometimes muscular tremblings and even epileptiform spasms may be observed. *Cattle* step suddenly back from their stall, leap wildly to and fro, bellow

loudly, butt with their horns, and often fall down with epileptiform spasms and twitchings, grinding of teeth, foaming at the mouth, and twistings of head, neck, eyes, etc. *Dogs* are extraordinarily restless and excited, howl and whimper, are given to biting, snap at flies, try to break loose, manifest uncontrollable movements, vomiting, twitchings and eclamptic spasms. With these psychic, sensory and motor evidences of excitement, the mucous membranes of the head are much redder, the pupils contracted, vision is disturbed, the eye often shining and fiery, and the ophthalmoscope reveals considerable active hyperæmia of the optic papilla; the temperature of the skull is raised, the pulse is full, its frequency increased and breathing quickened. In the stage of depression, which often soon succeeds, even in a quarter of an hour, the morbid aspect is completely reversed and has changed to a condition of stupefaction and dulness. The animal appears sleepy and inanimate, hangs its head, rests it on the bar or trough, has a staring, expressionless eye, takes no notice of its surroundings, strikes unnatural attitudes, and sometimes performs automatic movements (running round as in a circus, or pointing as though at game), or staggers and falls down in walking. Appetite is diminished, intermittent or unnatural, urination and evacuation are delayed; the pulse is at times below, and at others above, the normal; the pupil is dilated. The *course* of acute hyperæmia of the brain is very varied. Sometimes complete recovery takes place in a few minutes or, after repetition of the attacks, in a few hours or days. At other times an inflammation of the brain develops from the hyperæmia, or rather the latter forms the initial stage of the former; or it may end in fatal apoplexy due to vascular rupture or œdema of the brain. Lastly, abnormal conditions, such as sleepy staggers or vertigo, may remain permanently. The *prognosis* must therefore always be given with caution.

2. The symptoms of **passive hyperæmia of the brain** agree in the main with those of the second stage of the active form, and are chiefly those of depression. They are sometimes interrupted by signs of excitement. The morbid aspect is also frequently that of cerebral anæmia.

3. In acute cases of **anæmia of the brain** the symptoms consist in the occurrence of vertigo, which may increase even to insensibility (syncope) and apparent death. We notice weakness of heart, small pulse, vomiting (in carnivora

and omnivora), dilated pupils, paleness of the optic papilla (revealed by the ophthalmoscope). Sometimes the apparent death becomes real: so-called nervous stroke (apoplexia nervosa). But under some circumstances the aspect of anæmia of the brain may be altogether identical with that of acute hyperæmia, and present great excitement followed by stupor; this happens more frequently in cases of chronic anæmia of the brain.

Differential Diagnosis.—It is difficult to distinguish hyperæmia from inflammation of the brain, the symptoms of both being identical, and in many cases differentiation is quite impossible. In general, a slighter degree of intensity and a more favourable course, coupled with the absence (which is not invariable) of a feverish rise of temperature, point to such cases being hyperæmia rather than inflammation.

Therapeutics.—1. In addition to the removal of all causes of the disease, the treatment of *acute hyperæmia of the brain* demands an energetic attempt to prevent the excessive flow of blood to the brain. In this respect liberal blood-letting is the best remedy, if it can be done promptly at the outbreak of the disease; but later, when signs of depression have already appeared, it must be condemned. Besides this, the application of cold to the head is to be commended, and can best be done by an ice-bag, but must be occasionally suspended. If no ice be at hand, pour cold water on the skull. Finally, we attempt to divert the blood from the brain to the intestines by derivatives. Drastic remedies must, however, be used only at the very beginning of the attack, and for young and well-nourished animals. If the symptoms of excitement have passed away with the administration of drastics, then, according to our experience, the depressional stage will only be prolonged in consequence of too great a diversion of blood from the brain to the bowels, and the whole condition be thereby rendered worse. We therefore usually give only the milder laxatives, especially neutral salts and small doses of tartar emetic. Their operation may be assisted by cold clysters (infusions). Any derivation to the skin by irritant inunctions is rarely possible, owing to the timidity and excitability of the animal. Rather must all external irritants be withheld, and a quiet, dark, cool and airy room be provided. Give horses, if possible, a loose-box. Finally, see that cooling and laxative food is supplied; for horses the

best is green fodder, turnips and bran. Straw hats should be used for horses as a protection from the sun.

2. The treatment of *anæmia of the brain* is the contrary of that just specified. Especially during unconsciousness, which is its most frequent manifestation, the application of stimulants is imperative, viz., ether, alcohol, spirits of ammonia, camphor, coffee, caffein, hyoscyamine, atropine, strong skin-irritants, etc. In chronic anæmia of the brain, the general anæmia must be treated in accordance with its causes.

Kitt and Dexler emphasise the fact that the presence of dilated blood-vessels in the brain after death does not justify a diagnosis of pathological hyperæmia unless blood-spots and œdema are also present. Dexler is of opinion that abnormal variation of the quantity of blood in the brain is far more rare and attended by less significant results than is generally supposed. He doubts whether the accumulation of abnormally large quantities of gas in the bowels gives rise to hyperæmia of the brain, or whether anæmia of the brain may be brought about by the pressure of tumours upon the great arteries. He considers that the clinical appearances of cerebral congestion are so vague that diagnosis is practically impossible. He does not believe that the increased redness of the mucous membranes of the head should be attributed to the presence of an abnormal quantity of blood in the brain (?).

CEREBRAL HÆMORRHAGE. APOPLEXY OF THE BRAIN.

Etiology.—Apoplexy of the brain, also known as blood-stroke, stroke, brain-stroke, seizure, etc., consists in rupture of one of the larger or smaller vessels of the brain, with effusion of blood and compression or destruction of the brain substance. It is produced by various causes. First a cerebral hæmorrhage may occur in connection with hyperæmia or inflammation of the brain, so that the causes of the latter would have to be considered. In other cases vascular lesions form the starting-point of an apoplexy of the brain, *e.g.*, atheromatous and fatty degeneration of the cerebral arteries. These are, however, less frequently the causes in themselves of ruptures, than in conjunction with an increased blood-pressure. Traumatic influences, and especially shock to the brain, chiefly set up hæmorrhage in the cerebral membranes, particularly between the dura mater and the cranium (hæmatoma of the dura). Not unfrequently after emboli, hæmorrhagic stoppages occur in the brain, just as in other organs. Thus we observed one in a dog in connection with metastatic formation of carcinoma of the lungs. Finally, hæmorrhage on the brain takes place in

general disorders of a hæmorrhagic character, such as anthrax, petechial fever, distemper, the typhoid of birds, poisoning, and the intrusion of parasites into the brain (cercaria). Sudden death from cerebral hæmorrhage has been known to occur in old stallions during coition.

Post-mortem Conditions.—Cerebral hæmorrhage is most common in sheep, cattle and dogs; rare in horses. Its favourite localities in the brain are the vicinity of the lateral ventricles, the grey matter in the cerebral cortex, as well as the corpus striatum and the optic thalamus. Sometimes we find very small, so-called capillary, dotted hæmorrhages, and at others larger or apoplectic foci.

The *capillary hæmorrhages* are small, round or elongated blood-foci of a dark-red colour, varying in size from a millet-seed to a pea. The cut surface of the brain is sprinkled or streaked by them, and these dots cannot be wiped or washed off, thus differing from those which appear during a simple hyperæmia. If the discharged blood be still within the sheath of the pia of the vessels, these capillary hæmorrhages are termed miliary dissecting aneurysms.

The *apoplectic foci* attain to the size of a pea or hazel-nut even on the rupture of the smallest artery, but may reach very large dimensions according to the size of the vessel implicated, so that extensive areas of the brain-substance are often destroyed by pressure. The recently formed foci present a dark, black-red, coagulated mass, which is soft or pulpy, and the surrounding brain-matter is often also softened. In older foci the blood-coagulum is contracted and lighter in colour, and the adjoining tissue has been stained yellow by diffusion of the colouring matter of the blood. Later on, the extravasated blood and injured brain-matter are gradually re-absorbed, after previous decomposition, and either a smooth-walled, free space is left, which becomes filled with fluid (an apoplectic cyst), or the brain-substance undergoes shrinkage by formation of additional connective tissue (an apoplectic cicatrix), which is stained yellowish-brown by pigmentation from the colouring matter of the blood.

Symptoms.—The signs of cerebral hæmorrhage occur suddenly. They consist in stupor of the sensorium, vertigo, staggering and stumbling, trembling, involuntary movements (pressure to one side, running round in a circle, pointing, as

at game), unconsciousness, collapse, raging. Less frequently the psychic powers remain undisturbed. The mucous membranes of the head appear very red; in fact the hæmorrhage may extend to its tissues and outer surface, so that bleeding occurs from mouth and nose. The pulse is weak to the point of imperceptibility, breathing dyspnœic; and sometimes involuntary urination and evacuation may be noted. These signs may be complicated from the first or later on with paralytic symptoms, which, by their peculiarity, point to some local affection of the brain; so-called focal symptoms. Among these are paralysis of single muscles or groups of muscles (monoplegia), *e.g.*, one-sided paralysis of the muscles of lips, tongue, jaws, eyelids or ears, disablement of a hind or fore leg; further, semi-lateral paralysis (of the half of one side, or hemiplegia), paralysis of the optic nerve with subsequent amaurosis, paralysis of sensation throughout an entire half of the body (hemi-anæsthesia), paralysed deglutition (hæmorrhage in the medulla oblongata). Monoplegia indicates an affection of the motor centres of the cerebral cortex; hemiplegia points to hæmorrhage in the region of the path of conduction up to the pyramid, both upon opposite halves of the brain. A certain localisation of the local symptoms is often impossible. The *course* of apoplexy of the brain may be fatal in a few minutes or hours, but days and weeks may pass before the end, during which the attacks are sometimes repeated. Cure is rare and usually incomplete, paralysis remaining behind.

Diagnosis.—The recognition of cerebral hæmorrhage is based upon the sudden occurrence of severe cerebral attacks, complicated with signs of congestion of the brain and paralysis. In contrast to these, in so-called lung-stroke (extreme, active hyperæmia of the lungs with œdema), the symptoms of disturbed respiration are most prominent. The apoplectic form of anthrax, which presents the same phenomena as apoplexy of the brain, may be distinguished by microscopical examination of the blood. Finally, a brain-stroke may be confused with several forms of poisoning (prussic acid, strychnine). In such cases autopsy and chemical tests must decide.

Therapeutics.—The treatment of cerebral hæmorrhage is almost hopeless. Phlebotomy can be justified only during the stage of hyperæmia of the brain; later, it must be rejected as harmful. Cold applications after the congestion of the brain

has diminished are of little use. Something may, however, be attempted, as in brain-anæmia, with stimulating remedies, so as to relieve the depression of brain activity; for which give ether, alcohol and camphor. When the cerebral hyperæmia is of long duration, derivation to the bowels may be so far advisable as that it prevents hæmorrhage. The surviving paralyses should be treated with massage, electricity and with strychnine. Iodide of potassium may be administered internally, in order at least to try and hasten resorption of the extravasated blood.

STURDY OR STAGGERS (GID OR TURNSICK).

Occurrence.—Among parasitic diseases of the brain staggers is the most important. It is most common among sheep and, in the second place, cattle; rarely is it seen among horses, goats, antelopes, rabbits and hares.

Natural History Notes.—The cause of staggers must be sought in the presence within the brain of cœnurus cerebralis, the bladder-worm of the brain (staggers-worm, or brain-bladder); in rarer cases these latter are also found in the spinal medulla. As Küchenmeister first proved by experiment in 1853, cœnurus cerebralis represents the "nurse" of tænia cœnurus, a tape-worm which especially infests sheep-dogs and butchers' dogs. The sheep are infected by the proglottides or eggs contained in the dung of such dogs, which, dropped in the pastures, are picked up by them. As these eggs retain their vitality longer in wet seasons, the disease is more prevalent in such years. Sheep-dogs, on the other hand, are infected by eating the brains of sheep suffering from staggers. The development of the cœnurus-cysts occurs by liberation of the six-hooked embryo through dissolution of the tape-worm egg-shells in the sheep's stomach. The embryo then penetrates the membranes of stomach and bowels and wanders towards the brain, or spinal marrow; whether along the circulatory channels or by following the connective tissue is not yet decided. According to Müller, it is most likely that the brood reaches the brain by embolic methods through the blood. The certain proof of verminous emboli in the brain, and the fact that inflammatory changes are never found upon the basilary surface of the brain, seem to support this theory, especially as

such changes are common upon the upper surface of the hemispheres, and the dura is never found to be perforated. When once the embryos have settled in any part of brain or medulla, they lose their hooks and gradually change into bladders or cysts. After from 14 to 19 days they are the size of a millet or hemp-seed ; in from 26 to 42 days they reach that of a pea and in 50 are as large as hazel-nuts. They attain their organic development in from two to three months.

Post-mortem Conditions.—1. During the *emigration-stage* of the embryos into the brain, which represents clinically that of brain-irritation, we find on the upper surface of the cerebral hemispheres, in the reticulate tissue of the pia, round stripes, intensely yellow, about $\frac{1}{25}$ inch thick, which run sinuously upon the diagonal section. These, according to Möller, consist of a central hæmorrhage with a peripheral accumulation of pus-corpuscles, and therefore represent a localised hæmorrhagic purulent lepto-meningitis. Upon the corresponding place on the dura a similar fibrino-purulent pachy-meningitis is also present. These serpentine lines are nothing else than the immigration routes of the cœnuræ, at the terminus of which they may be found as cysts, from the size of a pin-head to that of a pea, usually several together (up to ten, Huzard says as many as thirty). Similar pathways are found in the brain itself in the form of winding passages, full of purulent, creamy fluid (encephalitis suppurativa). The embryos may even reach the choroid plesus, when suppurative hydrocephalus internus is set up, with swelling and suppurative infiltration of the plexus, as well as purulent proliferation on the part of the ependyma. Besides these, necrotic foci, $\frac{1}{8}$ to $\frac{3}{8}$ inch wide, occur in the brain, probably caused by verminous emboli. We find both in brain and plexus, hæmorrhages, small and large, and even extensive apoplectic foci.

2. In their *fully developed stage*, which is that of true staggers, we observe the cœnurus-cysts grown to the size of pigeons' or hens' eggs. Their shape varies from round to oval (in the spinal medulla a longer oval or even cylindrical), and they contain a thin fluid as clear as water. Their inner walls are covered with numerous scolices (up to 500), which may make them bulge outwards. There is either one such large cyst or bladder, or several (2 to 6) smaller ones, which displace the brain-substance and may even erode the bone of the cranium to the thinness of paper, so that it may be perforated. The brain imme-

diately adjacent is anæmic and sclerotic (atrophy of the brain). Very large cysts may occupy the entire half of a brain (Reboul). In addition to these changes in the brain, we may not unfrequently discover dead and calcified cœnuræ in the connective tissues of the body, in the subcutis, muscles, heart, liver, lungs, kidneys, mesentery, etc. These are granular and fattily-degenerated nodules, from a millet-seed to a pea in size. Fully developed cœnuræ have also been found under the skin.

Symptoms of Staggers in Sheep.—This disease, which is known by many local names, usually occurs in young sheep (lambs and yearlings), and is exceptional after their second year. In wet seasons especially it is almost epidemic and plays great havoc in a flock. Even newly-born lambs may have staggers, caused by embryos received from the mother during pregnancy. Three stages of the disease must be distinguished.

1. The stage of brain-irritation, following on the immigration of the embryos, the so-called "staggering." Time: from late summer to autumn; duration: from 8 to 10 days. 2. The stage of incubation, lasting from 3 to 6 months. 3. The stage of the true staggering sickness, commencing in winter and on towards spring, and lasting from 4 to 6 weeks.

1. The *stage of brain-irritation* is only observed, according to Möller, in about one-fifth of the animals which later have staggers, and its symptoms vary according to the extent of the immigration. Very often only trifling disturbances occur, such as melancholy and lassitude, a clumsy gait, stupid, expressionless look, or lagging behind the flock. In severer cases the brain-phenomena are much more intense. The mucous membranes of the eyes, especially the conjunctiva scleræ, are very red, the top of the skull feels hot, the animals hang their heads, are noticeable for great timidity and excitability, rush off suddenly or run aimlessly hither and thither, press to one side, run in a circle or turn round and round, are dizzy, stagger and easily fall, and show spasms and fits of twitching. This condition may end fatally in from 4 to 6 days with increase of stupor (according to Möller in 5 per cent. of all cases). In most cases, the symptoms abate from after 8 to 10 days, and in a few instances (not more than 2 per cent., says Zürn) complete recovery takes place. Generally, it is merely a prolonged pause in the disease.

2. The second *stage of incubation* usually passes without any noticeable symptoms. The animals are apparently healthy;

but on closer observation we may sometimes perceive a certain diminution in their intelligence.

3. The *stage of true staggers* commences with gradually increasing heaviness and stupor. The animal suddenly stands still, ceases unexpectedly to eat, is dull and faint, runs unconsciously against things, has a glassy, staring eye, with dilated pupil, and manifests peculiar characteristic motor-disturbances. The several unconscious and uncontrollable movements are as follows: (*a*) Running as in a circus, making smaller or larger circles round a central point in one or other direction; (*b*) twisting or pointing movement, in which only the fore or hind quarters are turned round a hind or fore leg, the head and neck being sunk; (*c*) the rarer rolling movement, in which the animal, lying on the ground, rolls round the long axis of its body; (*d*) in which it sways to one side, reels and often falls down—so-called "dizzy or tumbling staggers"; (*e*) in which, with lowered head and legs uplifted, it runs quickly straight before it—so-called "trotting staggers"; and (*f*) when it runs rapidly straight on, with the head raised or even bent to one side, frequently stumbling and even falling over backwards—so-called "sailing staggers." Of great diagnostic importance is the formation of a circumscribed area on the cranium, which is more yielding than the rest and even fluctuating. Pressure upon this generally causes pain and even convulsions. Unless the animals are soon killed or operated upon, death takes place in from four to six weeks from paralysis of the brain, accompanied by growing emaciation and feebleness. In isolated cases epileptic fits, strabismus and nystagmus have been observed.

Rump-staggers.—The immigration of cœnurus-cysts into the spinal medulla produces the symptoms of so-called "rump-staggers." These consist at first in weakness of the loins, swaying to and fro of the hind-quarters when walking; a gait resembling that of fowls, with the legs thrust under the body; pain and even collapse on pressing the flanks; and finally, complete paralysis of one or both hind legs, which are powerlessly dragged along. The animal perishes at last from general wasting and debility. Loss of consciousness, or any involuntary motions, are noticed only when there is a cyst also in the brain.

Symptoms of Staggers in Cattle.—Contrary to our experience with sheep, this malady is not rare in older animals among cattle, though still most common in yearlings. It begins with irregularity in the taking of food, the animal suddenly breaking off while feeding. Then timidity is shown, laziness in movement, together with a peculiar posture of the head, it being

lifted up and held sideways with a quick succession of twitching movements. The glance is staring and stupid, and the pupils are dilated. The root of the horns and the forehead feel hot; breathing and pulse are accelerated. There are disturbances of movement: the beasts press forward, and it is difficult or impossible to make them step back, or they show decided "circus" or rotary movements. Sometimes they fall down suddenly in their stalls, when they may even wrench off their horns, and show maniacal signs and spasms. Percussion of the skull should be carefully carried out, after previous removal of hair from the forehead, with the metal back of the percussion-hammer. Sometimes it detects a duller tone on the site of the cyst, and reveals great painfulness of the surrounding part. The result of the disease is the same as in sheep, but the course much more rapid. Compulsory slaughter is generally the rule, and, according to Rudolfsky, this is more necessary in the months of May and June. From the first symptoms to the climax of the disease four to five months may elapse.

Staggers in Horses.—This is exceedingly rare, and occurs at first with the aspect of sleepy staggers or inflammation of the brain. These signs are complicated with rotary movements around one leg, running in a circle, backing, dizziness, blindness, etc. Death sometimes occurs apoplectically.

Differential Diagnosis.—Staggers may be confounded with a number of other complaints and, especially at its outbreak, with cerebral inflammations arising from other causes, such as that produced by the larvæ of œstrus (œstrus ovis sometimes attains the brain); also with epilepsy, vertigo, blindness, suppurative catarrh of the antrum; and in addition to these cerebral tuberculosis in cattle and hydatid disease (echinococci). It is naturally impossible to distinguish it from these two last diseases during life; but it may be known from that caused by œstrus-larvæ, which is sufficiently characterised by the catarrhal processes on the part of the mucous membranes of the nose and frontal cavity. We may also exclude inflammation of the brain, in so far that it does not generally occur in the form of a focal disease. When once staggers has been shown to be present in a herd, the diagnosis of further cases is not difficult, and we will not go far wrong in taking every case of brain-disease in the same herd to be one of staggers. Confusion of rump-staggers with trotting-sickness need not occur, if we take into account the other distinguishing signs of the latter, and especially the so-called nibbling.

Prognosis.—The apparently favourable signs during the second stage of staggers are really a very unfavourable indication, because the disease, if uncombatted, is sure to end fatally, and because all the remedies we can apply are able to save only about one-third of those attacked. For this reason sheep which are bred for fattening had better be slaughtered at once. Opinions vary much as to the value of the treatment, which is purely operative; we share that of Dammann, Möller and others, that it can never prevent very heavy losses, and ought not therefore to be attempted whenever the disease appears in epidemic form.

Therapeutics.—Apart from prophylaxis, the treatment of staggers is essentially surgical and consists in removal of the cœnurus-cysts from the brain. This very old operation, known among shepherds as "staggers-boring," is more suited for sporadic than for enzoötic outbreaks. The reason why this plan succeeds in saving only about one-third of the affected animals lies in the fact that the cyst often cannot be located, or lies too deep for removal, and that in a third of the cases there are, not one, but several cysts. Moreover, hæmorrhage or suppurative inflammation may follow the operation, and in many cases the anatomical and functional disturbances of the brain have gone so far that nothing can save them. For a successful performance of the operation two factors are important: the localisation of the cyst and a suitable method of operation.

1. **General Diagnostic Indications for the Situation of the Cyst.**—If the location of the cyst cannot be fixed either by palpation or percussion, we may be guided by the nature of the motor-disturbances and by the position in which the head is held. Thus when it is carried sideways, that part which is lowest usually corresponds with the site of the cyst. In the course of time, and as a result of many comparisons of the clinical phenomena, certain diagnostic indications have been gained, but which, we must premise, are often misleading. The diagnosis in this respect can, therefore, only be one of probabilities. The several indications are as follows:

(*a*) In what we have called "*circus-movements*" the cyst lies superficially in the large cerebral hemispheres, and upon the side nearest the centre of the circle described by the animal.

(*b*) In right-handed "*pointer*" *movements*, either upon or

far within the right hemisphere, or else upon the floor of the left ventricle ; when left-handed then upon the reverse side.

(*c*) In "*trotting*" *staggers* at the anterior end of the hemisphere (frontal lobe).

(*d*) In "*dizzy*" *staggers* laterally in the lobe of the cerebellum or cerebrum.

(*e*) In "*sailing*" *staggers* between the cerebrum and cerebellum.

(*f*) In "*rolling*" *staggers* at the base of the cerebellum.

2. Of **methods of operation** two only can be considered, viz., trephining and use of the trocar. (*a*) For *trephining* sheep we use a crown about $\frac{1}{3}$ inch in diameter, and for cattle the instrument employed for horses ; the central line of the skull must f possible be avoided, owing to the longitudinal sinus beneath it. When the position of the cyst cannot be externally fixed, the chief spots for operation are : in male sheep, $\frac{1}{2}$ inch behind the middle of the horn ; in females, $\frac{2}{3}$ to $\frac{4}{5}$ inch behind the process of the horn, and the distance from the median line should be at least $\frac{1}{5}$ inch ; this spot corresponds with the centre of the posterior lobe. The spot immediately behind the inner corner of the horn or of its process (hinder part of the anterior lobe) is to be considered as only of secondary importance. In cattle, if the cyst cannot be located by percussion, the frontal cavity should be opened $1\frac{3}{5}$ inches from the iris and $\frac{4}{5}$ inch from the middle line. For the operation itself on sheep and cattle, see text-books of surgical instruction.

(*b*) The *trocar* should be used upon sheep so that the cyst may be pierced and emptied by suction, and then its wall may also be sucked out and removed. According to Zeden, the trocar is first inserted a finger-breadth behind the horns and the stiletto withdrawn, when a portion of the watery contents flows out, the rest being extracted by a suction syringe inserted through the cannula. The wall of the cyst is then drawn out by the syringe and pulled away with forceps. Dammann has modified this method, and inserts the trocar at first only $\frac{1}{3}$ inch deep, and pierces further when he fails in this way to reach the cyst ; moreover, the point of the trocar should be directed somewhat inwards. In case of need all the four spots named above and even the centre may be tried. Erdt's modification consists in using a punch for thicker skulls and introducing the trocar obliquely from the front inwards, a finger's breadth from the horn backwards and an equal distance from the median

line; the clefts of his cannula are serrated for better laying hold of and extracting the membrane of the cyst.

3. **Prophylaxis.**—Obviously more important than any surgical treatment is the prevention of the disease. This lies in destroying as far as possible the tape-worms of the sheep-dogs, which should be attended to several times a year; also in burning or boiling the brains of all sheep that suffer from staggers. In districts where the malady is prevalent, it would be well not to let lambs or yearlings pasture, but to feed them in the folds, in spite of any extra cost. The extirpation of other tape-worm harbouring animals, such as the fox, wolf and marten, has been proposed, but the suggestion is of only secondary importance.

Rolling-sickness in Dogs.—This may perhaps best be discussed in connection with staggers. It consists in fits of rolling which seize otherwise apparently healthy dogs, during which the head is at first held aslant and the whole body revolved several times around its longitudinal axis. There seem to be no disturbances of consciousness. The causes of this ailment, which evidently springs from an affection of the cerebellum, are various, but in general are little known. In a few cases we have found hæmorrhagic foci in the temporal lobe and on the crus cerebelli, which were of traumatic origin. Not unfrequently this rolling-sickness has been observed during nervous distemper and inflammation of the brain as an expression of some focal affection in the region of the cerebellum. In a solitary case we noticed the condition as a reflex phenomenon caused by constipation (relapsing). Mauri blames an embolus in the vessels of the cerebellum, having in two cases demonstrated a circumscribed softening of the same. As regards treatment, we obtained relatively the best results by administering sulfonal (8 to 30 grains), hypnon (4 to 30 grains) and urethan (½ to 5 drams). Among 70,000 dogs treated at the Berlin dog-hospital in nine years, only 33 suffered from rolling-sickness.

TUMOURS IN THE BRAIN.

Occurrence.—These tumours are rare, and appear most frequently in the choriod plexus in the form of chole-steatomata or margaritomata (which often produce no disturbance), melanomata, psammomata, fibromata and cysts, also as œdema proliferans. Also upon the cerebral membranes as sarcomata and lipomata of the dura, epitheliomata, dermoid and dental cysts; in the brain substance, as gliomata, glio-sarcomata and odontomata; and as exostoses, sarcomata and carcinomata of the cranial walls. With regard to tuberculous tumours, see the chapter on Tuberculosis respecting parasites; the para-

graphs on inflammation of the brain, sleepy staggers and staggers.

Symptoms.—The symptoms sometimes resemble those of general encephalitis, also those of sleepy staggers or apoplexy of the brain. Focal symptoms especially are a characteristic sign to a certain degree, but are often absent. As has frequently been observed in injuries to the brain, so in this case also the several parts of the brain react quite differently to tumours. If the tumours lie in the less important cerebral regions, such as the mesocephalon or corpus striatum, they may remain a long time without giving rise to any distinct symptoms. These are developed only when functionally important parts are attacked, such as the hemispheres of the cerebrum, with their psycho-motor centres, or the base of the brain with the places of exit of the twelve cerebral nerves. Such functional disturbances are usually restricted, owing to the limited nature of the tumour, and are known as focal symptoms. Without such focal symptoms, which gradually increase, and for which no internal or external cause can be traced, no opinion respecting the presence of a brain-tumour can well be formed during life, and a positive diagnosis is impossible. The following symptoms have been indicated in veterinary literature :—Twisting to one side, vertigo, disturbed power of balancing, epileptiform fits, psychic excitement, amaurosis, one-sided atrophy of the muscles of deglutition, hemiplegia, hemi-atrophy, hemi-anæsthesia, local muscular paralysis (monoplegia), etc. Of importance for diagnosis is the participation of the brain-nerves in the paralysis, viz., the nerves of the optic muscles, the visual nerves, the motor fibres of the trigeminus (masseteric nerves), of the facialis and hypoglossus. This indicates a basal position of the tumour. Respecting the occurrence in domestic animals of an engorged optic disc, such as is characteristic of brain-tumour in man, nothing is yet known. This is explained by the increased pressure within the brain caused by the tumour, whereby the cerebrospinal fluid presses into the lymph-sheaths of the opticus, thus giving rise to an œdema of the lamina cribrosa, with secondary compression of the papillary vessels and venous congestion in the papilla.

SUNSTROKE AND HEAT-APOPLEXY.

Definition and Nature.—Whereas until recently these two names were considered as almost synonymous, later researches have shown that the two conditions must be differentiated, since they represent separate affections. Although opinions are still divided as to the exact nature of both, yet the following may be laid down respecting their relation to each other.

1. **Sunstroke** (the *coup de soleil* of the French) is an affection of the brain caused by the direct action of the sun's rays upon the skull (brain and medulla oblongata). In slighter cases we have merely cerebral hyperæmia with its corresponding symptoms. In severer ones we meet either a paralysis of the vital centres in the medulla oblongata, especially the respiratory, or else inflammation of the brain or a sanguineous brain apoplexy.

2. **Heat-stroke or Heat-apoplexy** (*coup de chaleur* in French) is caused by over-heating of the body, combined with over-exertion and impeded outflow of bodily heat. It occurs mostly in horses which have severely exerted themselves during great heat, as in war or in manœuvres, also in pigs, sheep and cattle when driven in herds. Fat pigs, unaccustomed to transport, are specially susceptible. Many cases hitherto regarded as sunstroke evidently belong to this category. Respecting the nature of heat-stroke and the cause of death there is as yet no unanimity of opinion. Some authorities maintain that the chief cause is a change in the blood (destruction of the red blood-corpuscles, acid reaction of the blood, excess of carbonic acid, lactic acid or of urea, uræmic auto-intoxication). Others give it as a severe disturbance of innervation in the form of interrupted regulation of heat with abnormally high internal temperature and opaque swelling, especially of the cerebral ganglionic cells. (The temperature rises to 109.5° Fahr. and above.)

Symptoms.—1. In *sunstroke* the symptoms usually appear suddenly and without warning. In contrast to heat-stroke, there is no feverish rise of temperature at the beginning, nor often during the whole course. The symptoms differ according to the changes in the brain. Either we have the aspect of hyperæmia

or inflammation of the brain: restlessness, excitement, mania, spasms, trismus, vomiting; or sudden death occurs like apoplexy without any further signs; or the animals die with the phenomena of growing paralysis of respiration. In a case of sunstroke in a horse reported by Bartke, signs of restlessness, pawing, extreme redness of the mucous membranes of the head, violent mania and attempts to vomit were manifested; death occurring in three hours. Benjamin observed pronounced symptoms of rabies in a dog which had been exposed all day to a blazing sun. Siedamgrotzky saw a dog which lay chained up on a hot July day in the sun, and which suddenly died without any symptom. The upper surface of its brain was strongly injected and infiltrated with small hæmorrhages; between the dura and arachnoid lay a considerable quantity of bloody-serous fluid; and the cut surface of both brain and medulla oblongata was covered with numerous minute hæmorrhages. Another important point in differentiating sunstroke from heat-stroke is, that the former occurs without any previous exertion or movement.

2. The symptoms of *heat-stroke* are: weariness during work, profuse sweating, great rise of internal temperature, extreme difficulty of breathing, much timidity, palpitation, rapid pulse, which grows weak or imperceptible, at first dilation and then contraction of the pupil, reeling, staggering, swooning, collapse, trembling, twitching, jerking of the legs, with gradual approach of death. Recovery is rare. On *post-mortem* the blood is found of blackish-red colour and thick consistence, the muscles are dry and hyperæmia is present in the right heart, and especially in the lungs. If animals are subjected to severe exertion in hot sunshine, the symptoms of both diseases may be combined.

Between 1890 and 1895, 120 horses fell ill of heat-stroke in the Prussian army, of which number 115 (96 per cent.) died. Bartke has reported fully on several cases of heat-stroke in military horses, mostly occurring during the September manœuvres in hot, close weather and after severe exertion. All the sick animals showed great weariness; a few died before they could reach their stalls. Others staggered and fell in the stable, with very hard breathing and profuse sweating. In all of them palpitation was marked, and in some so violently as to shake the whole body and to be audible some distance away. The visible mucous membranes were dark red. Death was in many cases rapid and appeared to be due to asphyxia. The

internal temperature in those which recovered rose to 105° F., and remained an entire week at this point; they were not fit for service until after five weeks.

Differential Diagnosis.—For the differentiation between sunstroke and heat-stroke the most important points have been already given. But both maladies may be confounded with hyperæmia, inflammation and apoplexy of the brain; with vertigo, swooning, epilepsy, anthrax, some forms of erysipelas and of poisoning. In pigs the possibility of confusion with erysipelas is of special practical (forensic) importance, and a decision can be secured only by autopsy. In heat-stroke the changes shown are mostly negative, as compared with the very characteristic marks of erysipelas:—gastro-enteritis, swollen liver and spleen, nephritis and bacilli in the blood.

Therapeutics.—The treatment in both diseases consists in reduction of the heat (see that for inflammation of the brain), also in quiet, supply of water, and administration of stimulants, especially the subcutaneous injection of camphor, ether, alcohol, atropine, hyoscyamine, caffeine, veratrine, strychnine, etc. Quinine has also lately been recommended, and if the heart-beat be excessively rapid, chloroform. Straw hats, with a cold wet sponge or compress underneath, are recommended as a protective measure. Similar hats may be worn by dogs used in harness.

LIGHTNING-STROKE.

Occurrence.—Animals pasturing in the open, such as cattle, horses and sheep, are most liable to be struck by lightning; also military horses. The effects vary with the intensity of the flash and its direct or indirect action upon the several animals.

Symptoms.—The operation of a lightning-stroke either shatters the nervous system electrically, or burns the skin, or rends the softer parts of the body. Strong shocks kill instantly, as though by apoplexy, through paralysis of the nervous system. Weaker ones, or those which strike near at hand, produce various conditions of stupefaction and paralysis. Thus a state of stupor, such as we notice in cases of concussion of the brain, may remain for several hours. In other cases paralysis of single extremities is left behind (of an ear, of one side of the lower lip, of the swallowing muscles, or of an entire hind limb) in the form of paresis and paralysis. The paresis is marked by a

relatively more favourable course. Not unfrequently lightning causes external injuries. On the skin we may also observe striped or fork-shaped branching lines or figures, which indicate the path of the current, and are caused by dissolution of the hæmoglobin of the blood-vessels and its diffusion through the adjacent parts. Or the stream scorches up the prominent protective hair over a wide area, such as the eyebrows, forelock, etc. Along with this superficial singeing are deeper-lying burns of the cutis, sub-cutis and muscles, with laceration and blackening of the latter. In fatal cases there is sometimes hæmorrhage from the mouth and nose. A powerful electric current will produce similar results.

Post-mortem Conditions.—Beyond local changes the condition of a body killed by lightning exhibits little that is characteristic. The chief thing is a great fulness of the venous system, dark, thinly fluid blood, rapid decomposition of the carcase, incomplete rigor mortis and also small hæmorrhages in the inner organs (brain, kidneys, lungs, etc.), and beneath the serous membranes. Occasionally nothing at all abnormal is met with.

Therapeutics.—These are solely symptomatic. Stupefaction requires stimulants (camphor, spirit, ether, ammonia, veratrine, atropine, caffeine, hyoscyamine); and for the paralysis we apply skin-irritants (friction, massage, electricity, with sharp and stimulating embrocations).

CHAPTER XVI.

DISEASES OF THE SPINAL CORD AND OF ITS MEMBRANES.

Cerebro-spinal Meningitis. Spasms of the Neck.

Etiology.—Between diseases of the brain and of the spinal cord is spasm of the neck or cerebro-spinal meningitis, which might therefore just as well have been included among the former. As its name indicates, it is an inflammation of the membranes of the brain and spinal cord, though chiefly only of the anterior portion of the latter, and agrees perfectly, both in nature, symptoms and anatomical changes with spasm of the neck in man. The causes in our domestic animals are evidently to be sought in infection, as in by far the larger number of cases the malady appears enzoötically and even epizoötically. For this reason it might very well be treated among infectious diseases. We place it here, partly because little or nothing is known of the infective agent, and partly because it is so frequently mistaken for inflammation of the brain. The causes which were formerly generally assigned, especially in sporadic cases, such as chill, cold, bad, stormy, or raw weather, overheated stables, too early loss of coat, or food that is too rich, are all only of secondary importance.

We would here point out that of the many cases which are described in veterinary literature as cerebro-spinal fever, a large proportion do not belong to this category. This condition has been frequently confused with ordinary cerebral inflammation, apoplexy, the so-called Borna sickness of horses, and even with cases of poisoning. We have, therefore, based our description of the disease upon those cases only which approximate most nearly to the epidemic cerebro-spinal meningitis of man, and which cannot be confused with any other morbid condition, carefully excluding all cases of Borna sickness. According to the most recent investigations, this condition is not to be

regarded as a cerebro-spinal meningitis and will be described in a separate chapter.

Cerebro-Spinal Meningitis in Man.—According to Jäger and Weichselbaum, the epidemic cerebro-spinal meningitis of man is due to the agency of an intercellular diplococcus, the meningococcus intracellularis. Heubner, who first succeeded in demonstrating the presence of the coccus in the living subject, induced cerebro-spinal meningitis in two goats by the intradural injection of bouillon cultures of this micro-organism.

Occurrence.—Of our domestic animals, horses and sheep are most frequently attacked by this disease, then cattle, goats and dogs. It is especially younger animals that suffer, particularly lambs and yearlings, as well as young and vigorous horses; moreover, an enzoötic outbreak sometimes occurs in the spring. During such an epidemic both horses and cattle may suffer together. The disease is said to have been first observed in America during the early fifties of the last century, and was described by Large in New York. The earliest German notices date from about 1865. In 1876 the disease spread as a plague all over Egypt, and, according to Apostolides, 5,000 horses, 700 mules and 200 asses died in Cairo and its neighbourhood alone. Saxony, Silesia, Hungary and Russia have more recently had similar visitations.

Post-mortem Conditions.—The chief anatomical changes are at first a serous, and later a suppurative lepto-meningitis of the brain and commencement of the spinal cord. Consequently we find between the dura and pia mater of the brain, medulla oblongata and spinal cord, as well as in the ventricles of the brain, either a yellowish, turbid, serous fluid or a greyish yellow, milky, purulent and even fibrinous exudation, more especially on the basilary surface of the brain. Roloff also found in sheep a suppurative infiltration of the blood-vessels of the pia, particularly in their adventitia. Occasionally the projecting nerve-roots are discovered completely enveloped in purulent exudation. The upper surface of both brain and spinal marrow is œdematous and softened, and the external layer of the brain infiltrated with pus. We find also isolated foci of softening in brain and spinal medulla, and sometimes smaller or more extensive hæmorrhages in the membranes and

within both brain and spinal cord. The vessels, especially those of the pia, brain and spinal medulla, are greatly congested. With these signs, though not in all cases, general bodily changes have also been noticed, which point to the disease being of an infectious character. Among these are dark, discoloured, uncoagulated blood, a clay-coloured liver, opaque swelling and fatty degeneration of the internal organs, such as the heart, widespread hæmorrhage, catarrhal affection of the intestinal canal, particularly of the abomasum in lambs, etc.

Symptoms.—As in man, so also in the domestic animals, the clinical aspects of cerebro-spinal meningitis vary considerably with individuals; and not only with those of different orders, but with individual members of the same species. The signs of cerebro-spinal meningitis are a combination of those of brain inflammation and inflammation of the spinal cord, and consist mainly in stupor and somnolence (coma), signs of brain-irritation being rarer. These are complicated with fits of spasms in the muscles of head, neck and extremities, with a stiff, constrained attitude of the neck and a cramp-like twist backwards of both head and neck (opisthotonus); there is also generally a high infective fever. When the medulla oblongata is more especially attacked we often, though not always, perceive paralysis of the muscles of tongue and pharynx, with difficulty or even complete paralysis of swallowing (acute bulbar paralysis). This arises from affection of the motor nerve-centres. At times an attack begins with symptoms of acute gastro-intestinal catarrh, with slight colic. As in the case of man, the morbid aspect varies somewhat considerably with the intensity of the disease, according as the brain, medulla oblongata, or spinal cord be attacked, and according to the kind of animal.

1. In **horses** the disease begins sometimes with violent shivering, which may even shake the animal to and fro in its stall; at others with extreme heaviness and somnolence, so that it staggers or falls down; and in general the signs of a brain-inflammation are manifested, though mainly those of depression. In other cases slight colic and difficult swallowing are the signs first noticed; the animal makes repeated and vain attempts to swallow its accumulated saliva, which eventually flows from its mouth in long threads. In rarer instances the disease commences with opisthotonus. Later on we observe stiffness of the neck and pain on touching that

part; and in lifting the head or pushing the animal back, we notice a constrained posture of the neck or even of the entire spine, attacks of opisthotonus and trismus, heat of the head and neck, with marked prominence of the muscles of the latter, which are tight and hard to the touch. The animal grows more excitable, and suffers much from hyperæsthesia and timidity. Then spasms of the facial (the face looks as in tetanus) and shoulder-muscles appear, and also of the extremities, lasting for some minutes and followed by extreme relaxation, general muscular tremors and much debility. Clonic spasms of the lip and eye muscles are also observed (nystagmus). Paresis and even paralysis of the hind-quarters may occur later, with a tottering, clumsy gait and at last decided lumbar paralysis. Moreover, paralysis of the optic nerve (amaurosis), of the lips, ears, etc., may be seen. The visible mucous membranes are either much reddened or turn yellow; appetite is lessened or suspended, being very often complicated with difficult or paralysed deglutition and much secretion of saliva. The urine contains albumen and sometimes red blood-corpuscles; even pronounced hæmaturia has been observed. The temperature may rise to 106° Fahr.; but in some cases is hardly raised at all. The pulse seems sometimes to be very frequent, at others normal, or below, being generally small and feeble.

The *course* of the disease is acute; its average duration being from 8 to 14 days; but death may occur within from 3 to 5 days. It appears to be especially violent at the beginning of an epizoötic outbreak, when, indeed, sudden deaths have been noted, and the animals collapsed quickly in spasm (opisthotonus) and speedily died with the symptoms of general paralysis.

2. In **sheep** certain premonitions, such as melancholy, dejection, great dulness of the sensorium, salivation, sucking-spasms in the lips, rotary movements, etc., announce the disease itself. Soon afterwards the animals lie helpless on the ground, unable to rise, are frightened when touched and have various kinds of spasms. In particular the head and neck are twisted upwards and backwards so that both are in a line, or the head almost touches the withers. Chewing-spasms, grinding of the teeth, nodding of the head, with twitching and convulsions of the muscles of the extremities also set in. Brow and skull are hot, the conjunctivæ much redder, the pupils dilated. In lambs the temperature is often above the normal (possibly a fatal sign). The *course* is sometimes very acute,

so that death may occur in a few hours or days; sometimes the malady lasts from one to three weeks. The usual result is a fatal one, not only with lambs, but also with older animals. Thus Schmidt records 41 deaths among 43 sheep of various ages.

3. In **dogs** we perceive at first signs of great stupefaction; those of delirium are rarer. The skull feels hot, the animals tremble, are feverish, show difficulty of swallowing, knock against things, stagger and even perform involuntary actions (running in a circle, rolling). The muscles of the neck are hard and prominent, the neck itself is spasmodically drawn backwards, and there may even be general cramps over the whole body, broken only by short intervals, which lead eventually to exhaustion and death. It is said that 2 or 3 days before death the cornea is slightly dimmed. The average duration of the disease is 9 days (3 to 20); but it is very rare among dogs, only 10 cases being reported from the Berlin dog-hospital in 9 years, out of 70,000 treated.

4. In **cattle** the disease is accompanied by disturbance of appetite and rumination, restlessness, excitement, shaking and elevation of the head, stupor, stretching the muscles of the neck, attacks of trismus and opisthotonus, spasms in the thighs, twitching the lips, rolling the eyes and collapse when the head is lifted; finally a condition like paralysis sets in. The symptoms are the same also in *goats*.

Differential Diagnosis.—1. Cerebro-spinal meningitis in horses is most frequently mistaken for a simple *brain-inflammation*, which is easily done, as the latter is also accompanied by spasms and twitchings. A distinction can only be drawn between them when the signs of spinal affection are distinctly marked and become prominent. 2. With regard to the clinical distinction between cerebro-spinal meningitis and Borna sickness, cp. following chapter (negative *post-mortem* findings in Borna sickness). 3. A differentiation between cerebro-spinal meningitis and *tubercular basilar meningitis* is also not always easy; but is based upon proof of other tuberculous symptoms (tuberculosis of lungs or udder, emaciation), and upon the usual enzoötic appearance of cerebro-spinal meningitis. 4. *Fungoid poisoning*, especially as it occurs after eating ustilagineæ and mould-fungi, also gives rise to occasional mistakes.

Examination of the food supplies the key here. 5. In dogs, *rabies* and *distemper* have chiefly to be considered; but in by far the majority of cases a study of the collective symptoms will prevent any error. 6. In sheep confusion may arise with *cœnurus* or *staggers*, which also appears enzoötically, though the symptoms of the latter are essentially different, but autopsy is always the surest method of diagnosis. 7. *Tetanus* is distinguished from cerebro-spinal meningitis by the absence of abatement of the spasms, and by the cerebral symptoms.

Therapeutics.—The treatment of cerebro-spinal meningitis is in general the same as for inflammation of the brain. Ice-bandages or cold douches on the head must be applied first of all; derivation to the bowels by laxatives is also advisable. Then the anti-spasmodic remedies may be applied (subcutaneous morphia injections, bromide of potash, chloral hydrate, sulphonal, chloroform). Johne observed a favourable result in a horse after using pilocarpine (9 grains per dose). Whether treatment with lecithin as recommended by Fambach (7¾ grs. subcutaneously for horses) will prove of ultimate service, only time can show. From experiments undertaken in Prussian Saxony it hardly appears probable (see p. 685). A wise precaution would be to disinfect the stable on the enzoötic appearance of the malady and to remove at once all sound animals.

Progressive Bulbar Paralysis.—Under this name we understand in man atrophy of the motor nerve-nucleï of the medulla oblongata, especially those of the hypoglossus, facialis and vago-accessorius ("Bulbus" is the old name for the medulla oblongata), in the course of which paralysis and atrophy of the muscles of tongue, lips and jaws takes place, and also of those of the face, palate, larynx and pharynx, showing itself in difficulties of speech, mastication and deglutition, increased secretion of saliva, diminished reflex excitability, etc. Pneumonia (due to foreign bodies) is also noticed as a result of the impaired power of swallowing. Degive, Gérard, Laridon, Thomassen and others have observed a similar condition in horses in North-West Flanders. They reported a gradually increasing paralysis of the tongue, lip and jaw muscles, with disturbance of appetite, chewing and swallowing (the latter finally becoming impossible, food accumulating in the mouth or falling out of it, salivation), with emaciation, especially of the masticatory muscles, reduced reflex irritability, etc. The course of the complaint was very slow, death not taking place for from six to twelve months. Sometimes pneumonia also occurred, due to foreign bodies. Treatment was fruitless. The autopsy revealed discoloration, atrophy and fatty degeneration of the tongue and lip muscles, a slight loss of volume in the roots of the bulbar nerves; the medulla oblongata itself appeared normal. Under the name of "enzoötic bulbar paralysis of the horse," or "enzoötic spinal paralysis

of the horse," Thomassen describes a paralysis which occurs among horses in Flanders. It begins in the hind-quarters, ascends to the fore-quarters, and runs an acute course. As certain of the centres of the medulla showed pronounced degeneration of the ganglion cells, Thomassen classes the condition with the bulbar paralysis of man. He considers the cause to be poisoning by food (excess of turnips). Callum has described an "acute epizoötic leucoencephalitis" among horses in Maryland (America). The symptoms are: mental dulness, paralysis of the pharynx, staggering gait, and coma. Soft patches were found in the white substance of the cortex cerebri. An infectious paralysis of the brain and spinal cord was observed in twenty-four horses by Jacob. The symptoms were: difficulty in swallowing, weakness in the lumbar region, falling down, complete motor paralysis, and fever, consciousness being unimpaired, however, up to death. Fourteen horses died on the seventh or eighth day of illness. The autopsy showed inflammatory changes in the meninges. The cause of disease remained obscure. After thorough disinfection of the stables no further cases occurred. In the motor nucleï of man we find degeneration of the ganglionic cells with proliferation of connective tissue.

Epizoötic Spinal Meningitis in Cattle.—Whether the bovine disease described by Vogel and others under the name of "epizoötic spinal meningitis" is identical with, or related to, cerebrospinal fever is doubtful. The symptoms are identical: excessive salivation; difficulty in mastication and swallowing; restlessness; staggering gait; frequent lying down and getting up; paralysis of the lingual and pharyngeal muscles; rapidly increasing tendency to drop down; inability to get up; death on the third to fifth day. These symptoms do not point to anything more than an affection of the medulla. Psychic disturbances, muscular contractions and spasms have, on the other hand, never been observed. "Meningitis spinalis boum epizoötica" cannot be regarded, in any case, as a separate clinical entity, and it is probable that it has been confused with poisoning—especially mycotic poisoning—and with other pathological conditions. Thus, the case described by Zipperlen was, as he himself suggests, obviously one of mycotic intestinal inflammation, the *post-mortem* showing a high state of inflammation of the stomach and intestines (cp. chapter on Mycotic Inflammation of Bowels, p. 194). This condition, too, is accompanied by salivation, restlessness, paralysis of the muscles of the tongue and pharynx, marked tendency to fall down, etc.

Meningo-Encephalitis in Rabbits.—In two rabbits at the Children's Hospital of the Berlin Charité, Misch observed one-sided distortion of the head and vertebral column; abnormal focus of the eyes (so-called Magendie's position); incoordinate movements; one-sided paralysis; and nystagmus (rotatory). The autopsy showed abscesses in the left cerebellum and suppuration of the right cerebellar hemisphere. These processes contained specific diplococci, cultures of which, when injected intravenously into other animals, generally produced symptoms of disease similar to those shown by the original rabbits, in which the condition was spontaneous.

they appear as coffee-bean-shaped diplococci, but when transferred to artificial media, they develop into short streptococci with 6–9 rods. The rods are arranged both across and on the length of the chain, exactly as described by Johne in the diplococcus intracellularis equi, which he succeeded in isolating. When first propagated on artificial media the cocci do not do well. They soon become acclimatised, however, and flourish equally well on glycerine agar, common, or acid agar. Unlike the pus corpuscles, the Borna streptococci produce diffuse turbidity in bouillon. They may, however, be associated with the pus organisms in those cases where death from Borna sickness supervenes after several days of lying down. The Borna streptococci differ further from the pus bacilli in that they do not stain by Gram's method, and they are not experimentally pathogenetic. They do not liquefy gelatine. A good medium for the culture of the Borna streptococcus is also provided by water which has been contaminated, either by accident or design, with stable drainage, urine, or ammonia. The Borna streptococcus was first discovered in well-water which had become contaminated with the drainage from infected stables (Ostertag and Profé). The cocci thrive best at the temperature of the body, but they will also develop strongly, though slowly, at a temperature of about 60°. They die at once when dried, but may live as long as four months in damp sub-strata.

Borna sickness may be experimentally induced by the injection of pure cultures of the Borna streptococci under the dura mater from the foramen magnum, as practised by Johne (Ostertag). The same results may be obtained by the repeated injection at short intervals of pure cultures of cocci into the blood-stream (Profé). Single intravenous injections, or those which are repeated at longer intervals, will produce symptoms of Borna sickness in horses, but these will be only transient. The cocci disappear from the blood a short time after injection, and as, moreover, they are present only in very small numbers in the sub-dural and ventricular fluid of experimentally infected animals, the conclusion is inevitable that the pathological symptoms are caused by the decomposition of the streptococci in the blood and the consequent formation of free neural toxins. The Borna streptococci do not form soluble poisons, diffusable in the nutrient sub-strata.

It has not yet been found possible to induce the disease experimentally by introducing the agent in any other manner (by way of the sub-cutis, nares, eyes, ears, alimentary canal).

The lack of success with food experiments must not be taken as evidence against the possibility, under normal conditions, of infection by way of the alimentary canal. Similarly, in the case of swine-measles, the failure of the greater number of food experiments is not a proof that the disease germs are not conveyed to the animal in the fodder. It must be accepted as conclusive that the disease-producing streptococci are taken up by horses in their fodder (contaminated water), and that the disease is actually due to the decomposition of these streptococci in the blood. Why it is, however, that the disease attacks only single horses in small or large stables, and to what conditions its regular appearance in infected districts from January to August, and its gradual disappearance from August to December are due, are points upon which, as yet, we have no certain information.

Siedamgrotzky and Schlegel class Borna sickness as a serous cerebro-spinal meningitis. In 80 per cent. of their cases, they have succeeded in cultivating from the cerebro-spinal fluid a micro-organism, which appears usually as a monococcus, but occasionally as a diplococcus, and has been also found in clusters in the cell body. This coccus was shown to be pathogenetic to horses but not to mice and rabbits.

Johne, on the contrary, has in no case succeeded in establishing a serous cerebro-spinal meningitis or any other inflammatory condition of the brain and spinal cord. The fluid invariably present in the sub-dural spaces and the ventricles was undoubtedly an ordinary passive transudation and never partook of the nature of an exudation. According to Johne, Borna sickness is an auto-intoxication, brought about by the action of specific toxins upon the central nervous system. These toxins he considers due to the agency of a schizomycete, to which he gives the name of diplococcus intracellularis equi. These schizomycetes occur exclusively as small diplococci, which are found, in small numbers only, in fresh transudation and which resemble the gonococci in their peculiar shape—like a coffee-bean. They occasionally form tetrads, are inclined to form short chains, and show a remarkable similarity to the diplococcus intracellularis of human cerebro-spinal meningitis. They were found to be pathogenetic by inoculation to guinea-pigs, goats and horses. Intradural inoculation produced symptoms of cerebro-spinal meningitis in a horse.

Streit cultivated a micrococcus from an "infectious cerebro-spinal meningitis" among horses in Guelph (Ontario), which is remarkably similar to, and may be identical with, Ostertag's streptococcus. The disease ran a very acute course, the early symptoms being paralysis of the tongue and pharyngeal muscles.

Symptoms.—The initial symptoms of this very variable condition are general lassitude and indolence; loss of appetite; frequent gaping; and sometimes a slight attack of colic. These premonitory signs are accompanied by the typical symptoms of the disease, fibrillary twitchings of the face muscles (lips, nose,

working its way into the spinal cord, has been recorded. Rheumatic injuries have also been blamed. Inflammation may spread from some adjoining area and affect the spinal cord secondarily, *e.g.*, from a carious vertebra. A secondary inflammation of the spinal medulla may take place in various infective diseases, such as septicæmia, pyæmia, strangles, distemper, dourine, etc. Comény, Schmidt and others have reported upon a primary infectious spinal meningitis which broke out enzoötically among cavalry horses. Chronic inflammation of the spinal cord is most frequently observed among horses and dogs (so-called loin-weakness, ataxy, tabes dorsalis). Of 70,000 dogs treated in Berlin during 9 years, there were 96 cases of this ailment.

Anatomical Conditions.—The aspect agrees essentially with that found in brain-inflammation. In lepto-meningitis spinalis the pia mater and arachnoid are found hyperæmic, swollen and dull, infiltrated with ecchymoses, and with a serous (hydrorhachis) or purulent exudation between the spinal cord and the dura; the upper surface of the former being sometimes œdematously and suppuratively infiltrated. In traumatic inflammations this purulent exudation sometimes appears as an abscess and gives off a foul odour. The myelitis, that is, the inflammation of the spinal cord itself, is usually circumscribed and local, as in encephalitis, consisting of a purulent infiltration of the marrow with softening and hæmorrhage (red softening), whereby its substance is changed into a greyish-red, pulpy mass. Abscesses are less frequent. During a chronic course new growths of connective tissue are formed between the ganglionic cells and nerve-fibres, with atrophy of the nervous elements (myelitis interstitialis, or sclerosis of the spinal marrow). Pachy-meningitis spinalis is usually suppurative; during a chronic course of it small plates of cartilage and even of bone are sometimes found lodged in the dura mater spinalis, in the form of insular incrustations (pachy-meningitis chronica ossificans). More exact histological researches have been conducted by Dexler and Hamburger in dogs and horses suffering from spinal disease. Savary found ascending sclerosis of the spinal cord, together with chronic lepto-meningitis, in a horse suffering from ascending inflammation of the spinal cord, which terminated in paralysis and death. Liénaux saw a case of myelitis with formation of cavities (syringomyelitis) in a dog.

Symptoms.—The phenomena of inflammation of the spinal cord are easily explained by the functions of that organ. As is known, it forms the channel for the motor (anterior cornu) and sensory (posterior cornu) nerve-fibres of the trunk and extremities, and contains certain centres for the bladder, rectum and genital organs. In correspondence with these facts, inflammation of the cord is accompanied by the following symptoms :

1. *Disturbances of mobility*, accompanied by signs partly of irritation and partly of paralysis. The animals show a constrained gait, stiffness in the spine, twitchings and spasms, sometimes also permanent contractions of the extensors and adductors of the hind legs (spasmodic spinal paralysis); or they stagger in walking, tumble down and are either partially (paresis) or completely disabled (paralysis). In the latter case they cannot rise; larger animals lie altogether and can only be lifted up, while smaller ones drag themselves along (when only the hind part is affected), drawing the hind limbs like a dead appendage behind them (paralysis of hind limbs). No movement can be produced in them by strong electric shocks, or doses of strychnine sufficient otherwise to kill. When the lumbar medulla is affected, then only the hind legs and tail are involved, but when the cervical medulla is diseased, all four limbs are struck with paralysis. In the latter case the pupil also is sometimes contracted.

2. *Disturbances of sensation*, either in the form of intensified (hyperæsthesia) or of diminished or suspended sensibility of the skin-nerves (anæsthesia). The former manifests itself in pain, excitement and unrest when touched, especially near the spine. Smaller animals, such as dogs, yelp, larger ones shrink back, even try to bite, or rear wildly, as we observed once in a horse. Sometimes every movement is marked by signs of pain. The tendon-reflexes can be produced with striking ease (Westphal's sign). Thus, if we tap a dog quite gently with a somewhat jerky movement of the finger upon the patellar ligament, short, rapidly following flexor and extensor movements of the dog's leg will take place, with violent, convulsive twitching of the entire limb (Dexler). Paralysis of sensation is recognised by the loss of reflex sensibility to irritants, whether mechanical, thermal, or electric. Pinpricks, intense heat and strong electric currents are not felt.

3. *Disturbances in the bladder and rectum.* These commence usually with retention of urine and accumulation of fæces in

the rectum, resulting from paralysis of the detrusor urinæ and the muscles of the rectum. The collection of urine in the bladder leads afterwards to catarrh, pyelitis and even nephritis. Later we find the contrary, viz., involuntary passage of urine and fæces, due to paralysis of the sphincter vesicæ and ani (incontinentia urinæ et ulvi). In male animals we also sometimes observe continuous erection (priapismus) of the penis, followed by impotence and paralysis of this organ.

In addition to these symptoms we often find at first a higher temperature in the parts affected, followed afterwards by a lower; sometimes also œdematous swelling, outbreaks of sweating, and, in most cases of long duration, muscular atrophy. The prolonged lying of the animal soon brings on bed sores. The *course* is generally chronic, but the animals are usually slaughtered before the fatal end is reached. Cure is rare, or else there remains at least a permanent weakness of the loins and ataxy.

Differential Diagnosis.—Inflammation of the spinal cord seems to be most frequently confounded with rheumatic hæmoglobinæmia, which some look upon as an affection of the spinal medulla. The aspect of myelitis and meningitis spinalis as described in most text-books is therefore nothing else than the complaint formerly known as "black disury." The "rheumatic" paralysis of the hind limbs which appears during muscular rheumatism, and especially that form of it so prevalent in dogs as lumbago, is often mistaken for inflammation of the spinal cord; so are also spinal paralyses due to pressure by hæmorrhage and new growths, as well as the phenomena of paralysed hind-quarters which are sometimes seen in the course of inflammation of the kidneys, and which may be hard to distinguish. The last-named complaint may always be recognised by the characteristic change in the urine; as the rheumatic hæmoglobinæmia may also be known by the persistent hæmoglobinuria, the previous history of the case and its very acute course; and, finally, a muscular affection is distinguished by the absence of complete disablement, and the local painfulness of the muscles affected. The differential diagnosis of inflammation of the spinal cord is more difficult in the case of other diseases of that organ, especially hæmorrhage and new growths. In this respect all definite indications are often lacking, and autopsy alone can settle the question. In dogs,

however, the diagnosis of ossifying spinal meningitis is, as a rule, not difficult, the old animals gradually developing ataxia.

Therapeutics.—In recent cases the treatment of inflammation of the spinal cord has consisted in the application of cold or Priessnitz fomentations along the course of the spine. In many instances good results are obtained by the prompt use of skin-irritants in the form of blisters. For these we apply in the case of horses cantharides ointment, tincture of cantharides, croton oil or mustard oil; and for smaller animals cantharides-collodium is very good, as is also a salve of tartar emetic. Derivation to the bowels by laxatives is also advisable at the outset; and later, when paralytic symptoms become prominent, electricity may be usefully applied. For this purpose an intermittent (induction) current is generally used. We usually employ for dogs and small animals Spamer's apparatus, placing the electrodes in various positions at both ends or at each side of the spine, or alternately one at the toe-end of the disabled leg, the other in the middle of the spine. The strength of the current must be regulated by the degree of paralysis, and we should avoid giving too much pain. We have obtained improvement in numerous cases of chronic inflammation of the spinal cord by continuous electrification. For the motor paralysis strychnine may be administered internally. The subcutaneous dose of strychnine nitrate for a horse is from $\frac{3}{4}$ to $1\frac{1}{2}$ grains; for a cow, 1 to 2 grains; and for a dog, $\frac{1}{80}$ to $\frac{1}{20}$ of a grain. When the presence of a larger quantity of exudation in the spinal canal is suspected, pilocarpine may be tried (3 to 8 grains for a horse), and also arecoline ($\frac{3}{4}$ to $1\frac{1}{2}$ grains for a horse). In very obstinate cases cauterisation of the skin along the spine is recommended. Larger animals should also be placed betimes in slings, to prevent decubitus. Attention must moreover be regularly given to the emptying of bladder and rectum, by compression or use of the catheter in the former case, by hand in the latter. We may apply for the progressive atrophy of the muscles, and as a substitute for electricity, irritant embrocations of spirits of camphor or of mustard, oil of turpentine, liniments of ammonia, etc., and also massage.

Infectious Paralysis of the Spinal Cord (congestion) in Horses.—Under this name Comény has described an enzoötic among French cavalry horses. Its chief symptom was a paralysis of the hind-quarters, which ran both peracutely, acutely and subacutely, but only in the former case was conjoined with fever. Autopsy revealed congestive

hyperæmia of the membranes of the spinal cord, as well as hyperæmia, and softening of the cord itself. Investigations into the cause of the outbreak were fruitless; but its contagious character was clearly proved. Similar enzoötics (staggering from the loins; paraplegia; somnolence; general collapse; rapid emaciation of the croup muscles; death after 12–24 hours), arising from unexplained causes, have been observed in horses by Schmidt, Dieckerhoff, Lothes and Bongartz, Daweke, Jensen and Rasmussen, Dahlström, and the French veterinary authorities, Mulotte, Grangé, Mégnin and Blin. According to Dahlström, "infectious paraplegia of horses" has been known in Denmark for over fifty years by the name of "typhus of the spinal cord," and is there almost universally regarded as identical with hæmoglobinuria. It also occurs in Sweden (normal urine, negative post-mortem findings). Under the name of "slinger," Rasmussen describes a disease which occurs among foals in Denmark. It generally makes its appearance during the summer and autumn; is characterised by sudden onset accompanied by fever; and its symptoms are staggering gait, extreme weakness of the hind quarters and sometimes of the fore-quarters. The mental faculties are undisturbed. The course is chronic, and may extend over months or even years. Nothing is known respecting the cause; the autopsy shows hæmorrhages in the spinal cord. Zschokke has also observed the occurrence of enzoötic paresis in cattle, which he ascribes to infection by the bacillus coli.

HÆMORRHAGE AND NEW GROWTHS IN THE SPINAL CANAL.

Etiology.—Apart from the pressure exerted upon the spinal cord by an effusion of inflammatory fluid, compression may be caused by hæmorrhage on the one hand and by new growths on the other. Of the latter, which are very rare, the following have to be considered in our domestic animals: sarcoma of the dura (melanoma, spindle-celled sarcoma), lipoma, actino mycoma, glioma, myxoma, etc., and cœnurus-cysts. Moreover, exostosis of the vertebral column, disease of the inter-vertebral joints, ossifying pachy-meningitis, etc., may all lead to compression of the cord. Hæmorrhage is caused either by traumatic influences (fracture or shock to the spine, shot wounds, etc.), or by vascular disturbances, such as emboli. The blood is found either beneath the dura or the arachnoid or in the centre of the cord itself (hæmato-myelia).

Symptoms.—The pressure-paralyses caused by *hæmorrhage* in the membranes or cord are characterised by their sudden, apoplectiform occurrence (spinal apoplexy). By this they are distinguished from those arising from new growths, as well as from inflammation of the spinal cord. The history generally points to some traumatic cause, or examination will discover

one, *e.g.*, in dogs, great painfulness of the spine and lumbar region when bent or stretched. An improvement, or even recovery, may set in during the course of the paralysis, owing to resorption of the extravasated blood, which is never the case with new growths. Signs of irritation sometimes precede those of paralysis (twitchings, pain). Thus Kammerer observed a genuine attack of cerebro-spinal meningitis in a cow after hæmorrhage near the medulla oblongata. Schlesinger saw tetanic spasms of the extremities and muscles of the back in a dog, which quickly spread to those of the breast and neck, and also clonic convulsions of single groups of muscles, sensibility to pressure and stiffness in the spine, extreme hyperæsthesia of the skin and spontaneous evidences of pain.

Compressive *new growths* lead gradually to paralysis, and generally after previous symptoms of irritation. It is at first only partial (paresis), but later becomes complete (paralysis). Cure is impossible. The aspect of the malady closely resembles that of inflammation of the spinal cord.

Differential Diagnosis.—In addition to the above narrower distinction between the various spinal paralyses (caused by new growths, inflammatory and apoplectic), which is based upon their gradual or sudden appearance, the history of the case or its curability or the possibility of improvement—which distinction only too often cannot be made—it is particularly important from a practical point to be able to tell a spinal from a cerebral paralysis. We possess the following data to guide us in so doing.

1. Spinal paralyses are mainly paraplegia, and the cerebral are hemiplegia. The explanation lies in the fact that, owing to the smallness of the spinal cord as compared with the brain, the pathological process generally attacks it in its entire diameter. For the same reason the paralysis is both of motion and sensation (anterior and posterior horns or cornua); consequently all muscles situated behind the affected spot in the spine are paralysed.

2. In spinal paralysis there is usually no psychic disturbance, nor any participation of the cerebral nerves, which signs are mostly present in the cerebral form.

3. A simultaneous paralysis of bladder and rectum points to a spinal origin.

4. Spinal paralysis sometimes advances forward, and is therefore one which ascends.

5. Disturbed nourishment in the affected parts (muscular atrophy) is so far a characteristic of spinal paralysis as that the ganglionic cells of the motor anterior horns of the medulla possess a nutritive influence upon the muscles which they innervate. This is not to such an extent the case with the motor brain-centres. Muscular atrophy also occurs in cerebral paralysis (trigeminus).

Compressive paralysis in dogs is not unfrequently confused with a spinal paresis or paralysis caused by heart-disease (valvular defects). Thus we sometimes observe paralysis of the hind quarters during incipient dilatation of the heart following on valvular defects, which arises from venous hyperæmia (or arterial anæmia) of the medulla spinalis, or in other cases from thrombosis of the femoral arteries which has occurred in the course of such heart affection. In every case then of spinal paralysis in a dog it is well carefully to examine the heart.

Therapeutics.—The treatment of paralysis due to pressure is the same as for inflammation of the spinal cord. That from new growths is incurable. In hæmorrhage of the spine we might administer internal laxatives or iodoform to assist resorption.

Combined Paralysis of the Tail and Sphincters in the Horse (so-called "Sheep Tail").—Paralysis of the tail in horses, which is a recognised pathological condition and commonly known as "sheep-tail," has been recently investigated by Dexler. The disease occurs in mares, and is characterised by complete paralysis of the tail, of the sphincter muscles of the anus and bladder, and of the rectal muscles. It is, however, not to be ascribed to any known cause. According to Dexler the anatomical changes are: a chronic inflammation of the component parts of the cauda equina with very extensive new formation of extra-dural connective tissue. The clinical signs are: the drooping appearance of the tail, which swings slackly from side to side; the gaping of the uncontracted anus; the accumulation of fæces in the rectum, giving rise to attacks of colic; incontinence of urine; anæsthesia of the tail, root of the tail, and perineum, as well as of the mucous membranes of the rectum and vagina. The condition is incurable, but is not, in itself, dangerous to life, and the animals can generally be put to work. Treatment is purely symptomatic (the regular clearance by mechanical means of the fæces from the rectum).

Ataxy.—By "ataxy" we understand a disturbance of the co-ordination of movement, whereby all the muscles needed for a particular action are not set in motion at the right moment, but some either too soon or too late; or some groups of muscles alone are moved (such as the flexors) without any corresponding action of the extensors. One of the commonest ataxies is the "cock-stride" of horses, which may in many

cases be a result of spinal disease. Ataxic movements caused by an interstitial myelitis (pressure-atrophy of the nervous elements by excess of connective tissue) have been described in horses by Weber and Barrier. In one case of cock-stride Bolton found chronic degeneration of the lower dorsal and lumbar medulla on scattered spots and in the form of swelling, disintegration and diminished power of taking on stains in the axis-cylinders, disappearance of the medullary sheaths and a formation of interstices in the supporting substance.

TROTTING OR NIBBLING DISEASE IN SHEEP (*Trotters or Nibblers*).

Definition.—This disease is a chronic, non-febrile hereditary affection of the spinal cord, the nature of which has not yet been sufficiently investigated. It consists mainly in increased sensitiveness (hyperæsthesia) and in weakness and signs of paralysis in the hind quarters, with extreme emaciation, always ending in death. The most recent anatomical and bacteriological researches give only a negative result. In the spinal cord, more especially, investigations, both microscopic and macroscopic, have failed to prove any typical changes (Cassirer and Zuntz).

Occurrence.—The trotting-sickness was almost unknown before the introduction of the finer breeds of merinos (imported in 1765 from Spain), but has since become widely spread. It is particularly common in the very fine Electoral herds, while rarer among the commoner sheep of the country or the Negretti breed. The males sicken most readily, especially young rams from two to three years; females and wethers less frequently. This disease is one of the worst that can attack a flock, and is therefore much commoner in Prussia and Saxony, with their finer sheep-breeding establishments, than in South Germany.

Etiology.—The causes of trotting-sickness are first to be sought in heredity. It is usually introduced into a flock by a breeding ram, which is already visibly affected or else bears in it the germs of the disease. False principles of breeding exercise great influence; exaggerated refinement of the race by too little freshening with new blood, exclusive breeding between nearly related animals, pampering the young, unhealthy forcing, attempts to hasten maturity, the use of old ewes for breeding and the sexual over-excitement of males by too frequent and too early copulation. Whether, besides these causes, others, such as climatic or local influences (swampy, flooded pastures with too luxuriant and rich fodder, or very meagre, sandy soil), co-operate cannot at present be decided.

Symptoms.—The first stage of trotting-sickness opens with no important signs. The animals are very timorous, behave shyly and hurriedly, tremble when seized, have an unsteady, staring and stupid look, nod their heads, also bend them backwards and display a peculiar trembling motion of the ears—the latter especially when the sun is very hot. This stage lasts from 4 to 8 weeks in winter, but only from 8 to 14 days in summer.

The second stage has a winter duration of from 2 to 4 months, and in summer lasts as many weeks. It also begins gradually and with general slackness of the muscles and growing weakness in the hind limbs. The animals have an insecure, tripping or trotting gait (hence the name), in which they straddle with the hind feet and thrust them far under the body. Movements resembling the step of fowls are seen ("cock-stride"). The power of galloping and coition is lost. This ataxy is complicated later with very severe itch, especially in the lumbar region, which is betrayed by much gnawing and rubbing (hence the name of nibbling sickness). The animals bite themselves on the root of the tail, loins, back and also on the hind, and even the fore legs, producing bald, inflamed places upon the skin, or else the wool is rubbed off, becomes rough and shaggy. Paralytic signs, which travel forwards, appear during the later course in the hind limbs, the sheep stagger about when walking, tumble down, can scarcely rise, and grow very thin. Death then follows, preceded by increasing debility and exhaustion.

The clinical aspects of trotting-sickness have been recently described by Cassirer. The motor disturbance begins regularly in the hind-quarters. In the course of a few weeks it increases in intensity and spreads sometimes to the fore-quarters. No case of complete paralysis of the muscles of the affected extremities has been observed. The patellar reflexes were normal and the functions of the bladder and rectum were undisturbed. Tests by electric stimulus showed that sensibility was unimpaired. Death was the usual termination without complications. Autopsy rarely shed any light upon the actual cause of death. Cassirer does not believe that the motor protoneuron is the seat of disease (normal reflexes, sensibility to electric stimulus, absence of local muscular atrophy). There was no evidence that the peripheral sensory nerves were affected. The motor disturbances are ataxic in character, *i.e.*, due to derangement of co-ordination. There is, however, no clinical foundation for the conclusion that we have to deal with a condition similar to the tabes dorsalis of man. The fact that the reflexes are unimpaired militates strongly against this view. On the other hand, Cassirer considers that trotting-sickness in sheep does bear a certain resemblance to Westphal's pseudo-sclerosis in man, in which, similarly, the nervous system shows no evidence of pathological

change. Besnoit and Morel believe, however, that they have been able to recognise under the microscope definite changes in the spinal cord and in the peripheral nerves (degeneration of single large cells in the anterior horns; peripheral neuritis).

Differential Diagnosis.—This disease may be confused with rump-staggers (cœnurus-cysts in the spinal cord) and other spinal paralyses. A distinction must be based upon the symptom which is particularly important in trotting-sickness, viz., the presence of timidity and nibbling. The occurrence of the disease chiefly among male animals and in finer breeds is also evidence of trotting-sickness.

Therapeutics.—Any treatment of the sick animals is useless, and it is best to slaughter them early. Much more important is prophylaxis, which consists in preventing all affected animals from breeding, in not using the rams until after their second year and avoiding any sexual over-excitement; also in natural methods of keeping and feeding, especially of the young. It is also well to avoid too great a refinement of race by inbreeding.

Tabes dorsalis.—Under the names of tabes dorsalis, sclerosis of the posterior spinal processes, or progressive motor-ataxy, we describe in human medicine a chronic affection of the spinal cord accompanied by signs of paralysis, from unknown causes. The former erroneous opinion traced them to sexual excess. It seems that severe chill and great over-exertion of the legs, on the one hand, and an inherited predisposition on the other, are the most important sources of the disease. In regard to the latter point, it has been noticed that several members of the same family have suffered from tabes, which attacks men most frequently of from thirty to fifty years. The malady consists *anatomically* in grey degeneration of the hinder processes of the spinal cord, with extreme proliferation of connective tissue (sclerosis). The *symptoms* are in the main those of disturbed co-ordination of muscular movement (ataxy). They begin with signs of spinal irritation, shown in hyperæsthesia of the skin and in keen and darting neuralgic pains in the arms and legs, which stage may last for years. The ailment is often confused with rheumatism, ischias and other independent neuralgias. The second and so-called manifest stage consists in derangement of co-ordinated motion (motor-ataxy). It begins in lassitude, in a certain insecurity and awkwardness of gait, whereby the feet are often lifted unnecessarily high, moved backwards or set down with a stamp (" cockstride "). Sensibility is at the same time weakened, so that the resistance of the ground cannot be felt (" walking on clouds "). The third, or paralytic or paraplegic, stage is that of complete spinal paralysis.

Does this tabes dorsalis of man exist among animals? This question can only be tentatively answered. The trotting-sickness of sheep, for

instance, is very like it. Compare their heritability, their occurrence chiefly in males, their two stages of irritation and paralysis and the undoubted motor-ataxy. We believe also that in treating dogs for ataxy we have several times had true cases of tabes dorsalis before us, but have made no pathological investigation. Hamburger, on the contrary, proved decided tabes-like degeneration of the posterior processes in a dog, without, however, having seen the dog alive. The proof of tabes dorsalis in domestic animals during life is difficult, because the subjective symptoms (hyperæsthesia, neuralgia, loss of feeling) are not usually well marked. It is much to be desired that more exact pathological investigation should be made, combined with observation of the symptoms during life, viz., in sheep (trotting-sickness), in dogs (ataxy) and in horses (cock-stride, lumbar weakness).

CHAPTER XVII.

DISEASES OF THE PERIPHERAL NERVES.

PARALYSIS OF INDIVIDUAL PERIPHERAL NERVES.

I. FACIAL PARALYSIS.

Origin.—The causes of facial paralysis (paralysis nervi facialis) are very varied. The condition may be due to an affection of the facial centre in the brain (central facial paralysis), or to an attack of the peripheral portion of the nerve at the point where it bends round the inferior maxilla (peripheral facial paralysis). Central facial paralysis may affect either one side (central monoplegia), or both (central diplegia). Peripheral facial paralysis is generally one-sided (peripheral monoplegia); and is the form most frequently encountered. The causes of central facial paralysis, which is a rare form, are new growths, abscesses, hæmorrhages, thrombosis, inflammatory conditions and toxic processes (infective pleuro-pneumonia, glanders, *maladie du coit*) in the brain, giving rise to central diplegia. Or it may be due to a pathological condition of the portion of the facial nerve which lies between the floor of the brain and the bend at the inferior maxilla (sub-parotid melanosarcoma, affections of the petrous portion of the temporal bone, diseases of the ear, dental cysts). The causes of peripheral facial paralysis (common form) are principally contusion of the facial nerve at the inferior maxillary bend, due to pressure by the halter (left buckle!); pressure from lying down (colic, severe illness, parturition). In rare cases, injuries (lance-thrusts) and chill may also give rise to peripheral facial monoplegia.

Symptoms.—The symptoms and course vary according to whether the paralysis is peripheral or central.

(*a*) Peripheral facial paralysis is generally confined to one side only, and is traumatic in its origin. It shows itself as a one-sided paralysis of the lips (crooked mouth, drawn towards the unaffected side; difficulty in taking food and water); of the nose (contraction of the nostril, nasal dyspnœa); and of the

cheeks (difficulty in mastication, the food becoming lodged between the paralysed cheek and the back teeth). The course of one-sided peripheral facial paralysis is generally favourable, the symptoms frequently disappearing spontaneously in about a month. Double peripheral facial paralysis runs a less favourable course, both nostrils being contracted. There is pronounced dyspnœa, which, after rapid movement and certain other forms of exertion, may lead to suffocation.

(*b*) Central facial paralysis is characterised by paralysis of the muscles of the eye and ear, in addition to those of the lips, nose and cheeks. This form may affect either one side or both. The paralysis of the eyelids shows itself in two ways. The orbicularis palpebrarum, the muscle surrounding the eyelids, becomes paralysed, and the eyelids cannot in consequence be closed (lagophthalmus); while the corrugator supercilii, the external elevator of the upper eyelid, droops over the eye (ptosis). The implication of the muscles of the ear is shown by the drooping of one or both ears. The prognosis in central facial paralysis is far less favourable than in the peripheral form. When the condition is due to central tumours, it may be regarded as incurable.

Treatment.—Massage, rubbing with stimulants (spirits of camphor, oil of turpentine), electricity, and the sub-cutaneous injection of strychnine and veratrine promote the restoration of nerve conduction. The nasal dyspnœa may be relieved by raising the sunken nostrils by means of sutures. Under certain conditions tracheotomy may have to be performed.

2. PARALYSIS OF THE TRIGEMINUS NERVE.

(*Paralysis of the muscles of mastication.*)

Causes.—Peculiar paralytic conditions appear sometimes in the domestic animals, especially in the dog, due to affection of the third motor branch of the trigeminal nerve (ramus mandibularis s. inframaxillaris), which supplies the muscles of mastication. These affections are called variously trigeminal paralysis, paralysis of the muscles of mastication, masticatory paralysis of the face, and inferior maxillary paralysis. As in the case of facial paralysis the causes may be either central, *i.e.*, in the brain, or superficial.

(*a*) Central paralysis of the muscles of mastication is caused by diseases of the brain. Of these, the most important is rabies, which is always accompanied, in dogs, by paralysis of the lower jaw. The condition also occurs in the cerebral form of distemper. It may be also due to other pathological conditions, such as abscesses, hæmorrhages, and new growths (sarcoma).

(*b*) Peripheral paralysis of the muscles of mastication is either traumatic or rheumatic in its origin and occurs in isolated cases in dogs.

Symptoms.—Affection of the motor division of the trigeminus is shown by the passive dropping of the lower jaw, the animal being unable to close its mouth. It is quite easy, however, to raise the lower jaw with the hand, and the condition may be distinguished in this manner from dislocation. There is also a considerable difficulty in the prehension and mastication of food. Should the paralysis be of long duration (distemper), there may be considerable atrophy of the masseter and temporal muscles. Where the paralysis is the result of rabies, the condition terminates fatally within a few days, but in distemper and in the peripheral form it may last for weeks, or even months. Although the prognosis is in general unfavourable, recovery occurs in rare cases (massage, electricity, strychnine injections).

3. PARALYSIS OF THE SUPRASCAPULAR NERVE.

Causes.—Paralysis of the suprascapular nerve is peculiar to horses, and is brought about by traumatic influences (collision, slipping, falling, parturition, fracture of the shoulder-blade), implying a violent backward movement of the shoulder, by which bruising, crushing or tearing of the nerve, at the point where it bends round the anterior edge of the shoulder-blade and comes near to the surface, is occasioned. The suprascapular nerve springs from the brachial plexus. It bends round from the inner side of the shoulder-blade, between the subscapular and supraspinatus muscles, and reaches the periphery two finger-breadths above the scapular prominence, whence it supplies the two spinata muscles and the deltoid.

Symptoms.—Suprascapular paralysis shows itself by a peculiar abduction of the shoulder-joint as soon as weight is put upon it (so-called "slipping"). The spinata muscles and the

deltoid, which are supplied by the suprascapular nerve, normally serve as lateral supports for the shoulder-joint and prevent its giving way when weight is put upon it. Paralysis of the nerve interferes with this function and the shoulder gives way outwards (" slipping "). This causes a well-defined space to appear between the shoulder and the thorax (frequently seen in cattle). Atrophy of the paralysed muscles occurs after 2–3 weeks. This atrophy of the spinata muscles and of the deltoid is to be regarded as a degenerative process, and is usually very pronounced, causing a flattened or concave appearance of the muscles.

Prognosis is, as a rule, unfavourable, an average of about 75 per cent. of the cases being either incurable or only to be cured after several months of treatment, though Schimmel actually maintains that every case is curable. Particularly unfavourable for the prognosis is the development of pronounced atrophy of the anterior spinata muscle. Apart from the very slight form which disappears in a day or two or a week or two, recovery may take place under favourable conditions in 2–3 months.

Treatment.—This consists in massage (tapotement), electricity, rubbing with stimulants and the subcutaneous injection of veratrine or strychnine ($\frac{1}{3}$–$\frac{3}{4}$ grs.). Schimmel particularly advises the systematic movement of the affected limb.

4. RADIAL PARALYSIS (SO-CALLED).

(Paralysis of the anconeus muscle.)

Description and Causes.—A number of obviously dissimilar morbid conditions have been hitherto included in the term "radial paralysis," and it is important to distinguish between them.

(*a*) A neurogenic paralysis of the elbow extensor (triceps extensor brachii) and of the extensor longus digitorum pedis occurs in the horse, and, more rarely, in the dog and the ox. It is the result of peripheral paralysis of the radial nerve, due to mechanical injury (contusion of the shoulder after a fall, lying down, parturition, tying up to the rack, as the result of a kick, of a blow from the shafts, or of striking against a door-post). The curve of the radial nerve round the humerus at the

left condyle is a point particularly exposed to such injury. Contusions at this spot give rise to paralysis and atrophy of the extensor pedis (Lutz).

(*b*) A myogenic paralysis in the horse is sometimes occasioned by parenchymatous myositis of the elbow extensor (over straining of the muscles during parturition, hæmoglobinæmia).

Symptoms.—The function of the extensor brachii is to extend the elbow-joint. If these muscles, or the radial nerve which supplies them, become completely paralysed, the whole leg gives way when weight is put upon it. This total paralysis differs from partial paralysis or paresis, in that pressure upon the joint in the latter condition gives rise only to unsteadiness and frequent stumbling. Where the affection remains unchecked, atrophy of the extensor muscles may develop.

Prognosis.—Radial paralysis permits of a more favourable prognosis than suprascapular paralysis. In the greater number of cases, recovery takes place in about a month. Mild attacks (paresis) may recover in a few days.

Treatment.—As in the case of suprascapular paralysis, massage, electricity, rubbing with stimulants, systematic movement, and strychnine and veratrine are all indicated.

5. PARALYSIS OF THE QUADRICEPS MUSCLES.

(So-called crural paralysis.)

Description and Causes.—Up to the present, the name of " crural paralysis " has been given to a peculiar affection of the patellar extensor (quadriceps femoris) in the horse, which is supposed to be due to a paralysis of the crural nerve, an assumption which is not, however, justified by facts. In a small proportion of the cases only is the affection neurogenic in its origin. In the great majority of instances we have to deal with a myogenic paralysis of the quadriceps itself. Thus, two different forms of quadriceps paralysis, due to two separate causes, are to be recognised, namely, the myogenic form and the neurogenic form.

(*a*) Myogenic paralysis of the patellar extensor is brought about by a parenchymatous myositis which is usually the result of hæmoglobinæmia. In very heavy draught horses the con-

dition sometimes locates itself, either entirely or with special intensity, in the patellar extensors. The autopsy shows degenerative inflammatory changes in the affected muscles (light grey colour; softness; granular clouding; fatty and hyaline degeneration; absence of striation). Similar extreme muscular changes, with paralysis of the quadriceps, are occasionally observed in heavy horses as a result of over-straining the patellar extensors during parturition by pressing against the pasterns; of over-straining by kicking, slipping and falling (nervous over-strain, analogy with paralysis of the extensor brachii).

(*b*) Neurogenic quadriceps paralysis is the result of crushing or tearing (inflammation ?) of the crural nerve. It occurs but rarely.

Symptoms.—The most important symptom of paralysis of the patellar extensor is a remarkable one-sided lameness of the supporting leg in locomotion, which manifests itself in complete collapse of the entire leg from the stifle joint when weight is put upon it. There are no other signs. A degenerative muscular atrophy soon appears, the affected muscle gradually becoming slack, thin, aponeurotic, and shrunken. The course is, in the greater number of cases, chronic. The condition frequently lasts unchanged for several months, the horse having to be finally slaughtered. In exceptional cases recovery may take place after six months. Only in the minority of instances (about 30 per cent.) is recovery rapid. For this reason the prognosis is very unfavourable (the losses amounting to 70 per cent.). It is particularly so in double quadriceps paralysis, the animals often dying in the course of a few days as the result of decubitus.

Therapeutics.—Treatment consists in massage; rubbing with stimulants, electricity (faradisation; electric treatment is well borne by most horses after a time); and the sub-cutaneous injection of caffein, veratrine and strychnine. The animals should be systematically exercised when able to move.

Paralysis of the Brachial Plexus is observed occasionally in dogs and horses after serious injury, contusion, distortion and concussion of the shoulder; in fracture of the scapula; and in the course of sub-scapular phlegmon, inflammation, generally purulent, of the connective tissue. It consists in a complete motor and sensory paralysis of the entire limb. Muscular atrophy soon follows. Prognosis is unfavourable, the greater number of cases being incurable. Treatment consists in massage, electricity, and rubbing with stimulants.

Paralysis of the Ischiatic Nerve (great sciatic nerve).—Sometimes seen in dogs as the result of traumatic influences ; occurs also in the course of distemper. It is met with, but more rarely, in the horse as a result of abscesses in the glands of the pelvis. The paralysis affects the flexor longus digitorum pedis (tibial nerve) in such a way that the limb cannot be flexed, but hangs loosely down. It also attacks the extensor longus digitorum pedis and the tibial extensor (peroneal nerve), with the result that the pastern knuckles over when weight is put upon it. There is also anæsthesia.

Paralysis of the Sacral Plexus.—In addition to symptoms characteristic of paralysis of the ischiatic nerve (see above), there is paralysis and one-sided atrophy of the gluteal muscles, of the biceps and semi-tendinosis, together with anæsthesia of the muscles of the croup.

Paralysis of the Obturator means also paralysis and atrophy of the adductors of the thigh (gracilis, sartorius, pectineus, obturator, adductors), with abduction of the leg and incapability to move it inwards.

Neuralgias, which are paroxysms of pain in the region of distribution of any particular nerve-branch, have not yet been observed in our domestic animals, but probably only because they lack the subjective power of conveying their sensations to us. In man they are somewhat common, and attack especially the trigeminus, occipitalis major, the brachial and lumbar plexi, the intercostal nerves and the ischiadicus (neuralgia of the latter is called "ischias"). Friedberger noticed in a horse a peculiar and exceedingly severe itching on the skin of the neck and forearms, for which no local causes could be discovered, but which was considerably relieved by sub-cutaneous morphia-injections. Whether this evident neurosis of sensation was of neuralgic nature or is to be looked upon as corresponding to the pruritus of man, cannot be decided. Strebel and Martin have described similar cases in cattle. Serini and Giovanni believe that they have seen ischias in cattle and dogs, and a neuralgia of the brachial plexus has been observed by Catoni in a horse. Trigeminal neuralgia or neuritis of the maxillary nerve (branch of the trigeminus which supplies the upper jaw) is supposed to give rise to a characteristic swinging to-and-fro movement of the head in the horse, with continued rubbing of the nose and lips against hard objects. It is said to be curable by the excision of the infraorbital nerves on both sides (Williams, Liautard).

Facial Spasms (*Tic convulsif*) in a dog, and in the form in which they occur in man, have been described by Cadiot, Gilbert and Roger. Similar seizures have been observed by Bartolucci and Dexler in the horse.

Spasms of the Accessorius in a horse are described by Aruch. The papillary muscles of the lower jaw and the inferior cervical muscles are affected. The symptoms are: backward jerking of the head and neck, nodding, and lateral movements.

Hypnotism (auto-suggestion) in the horse is described by Zschokke.

CHAPTER XVIII.

NEUROSES WITHOUT KNOWN ANATOMICAL BASIS.

EPILEPSY.

Definition.—Epilepsy is not common among our domestic animals, but occurs in all, including poultry. It is a chronic brain affection with fits of disturbed consciousness and sensation and accompanying muscular spasms, between which are free intervals of varying periods. Regarding the nature and seat of the disease recent investigations have procured us some knowledge, which, however, is far from complete. The seat of epilepsy is especially to be sought in the motor centres of the cortex of the cerebrum. The chief experimental proof of this theory (the cortical theory) lies in the fact that by irritating the motor cortex-areas of the cerebrum, epileptiform fits can be produced in animals, and that, after such a centre has been extirpated, the spasms connected with its group of muscles cease. Moreover, the spasms in epilepsy correspond entirely with those of the muscular groups innervated by the motor-centre in question, and epileptic fits have been noticed in man after pathological affection of the cerebral cortex. But, above all, the invariable complication of the spasms with unconsciousness indicates clearly the cortex as the seat of origin of the malady. Bergmann has shown that such injuries to the skull as affect more especially the cerebral cortex produce both epileptiform and true epileptic fits. The experiments of Hitzig, Fritzsch, Luccani and others have proved that such fits may be caused by electric irritation applied to any part of the cerebral cortex, but that in the motor region the very faintest shock suffices. Moreover, after removal of this motor region, no attacks can be produced electrically from any portion of the cerebral cortex; but, on the other hand, they can still be produced even though the entire cortex be removed excepting that of the motor region. Unverricht found that the cortex

is not only the starting-point of epileptic attacks, but that it is also responsible for their continuance and development. Whether in addition to the cortex of the cerebrum, more deep-seated portions of the brain are the starting-point of epilepsy (infra-cortical or medullary theory) appears doubtful.

What the exact changes in the cerebral cortex are, is as yet unknown. Anæmia of the brain caused by sudden vascular spasm was formerly suspected (Kussmaul and Tenner); but other observers could not demonstrate the presence of such an anæmia. We must, therefore, assume a peculiar fleeting state of excitement in the cortex of the cerebrum, and especially of its motor cortex-centres, as the producing cause of epilepsy, which is perhaps brought on by an abnormal chemical or molecular composition of the substance of the cerebral cortex (Rosenbach).

Etiology.—In practice it is well to distinguish as regards their causes several varieties of epilepsy.

1. *True genuine idiopathic or spontaneous epilepsy* is a purely functional neurosis of the brain, without any discoverable pathological change. Its essential causes are not known. Among those which produce a predisposition thereto, heredity plays a leading part, as has been established by various authorities. Thus La Notte noticed the frequency of these fits in a herd descended from two epileptic bulls; they usually attacked cows after their first calf, and oxen when they were set to work. For this reason epilepsy is regarded as a "hereditary taint" in breeding paddocks. It is possible also that a bad condition of nourishment, rickets, the pampering of young animals, etc., may all predispose thereto; breeding stallions are particularly subject to attack. Out of 70,000 dogs treated during nine years in the Berlin dog-hospital there were 419 cases of epilepsy; on the other hand, it occurred only three times in the years 1891–2 among 30,000 service horses of the Prussian army, and in the years 1890–5 there were only 12 cases in all.

Among accidental causes which may produce epileptic fits in domestic animals when the predisposition already exists, experience has taught us to recognise several. As in man, so here also, certain psychic emotions stand in the first place, such as fright, excitement, dread. Thus it is reported that a fourteen-year-old gelding, trained by its owner, had its first fit when ploughing, on being startled by a suddenly thrown white cloth. In another horse the cause was a violent fright occasioned by a

stork unexpectedly flying up; a dog had a fit as a locomotive rapidly dashed past. In horses epilepsy often arises from glaring rays of light acting on the eyes, or from too sudden changes from light or darkness, thus, for instance, in driving rapidly towards the sun when low down on the horizon, or in an avenue when the setting sun throws the tree-shadows diagonally across the road, also in a strong reflected light from surfaces of water or snow. With many horses, the use of blinkers may give rise to epilepsy.

2. *Symptomatic epilepsy*, in contrast to the genuine idiopathic form, is produced by certain pathological, chronic, morbid conditions of the brain and its membranes, such as new growths, parasites (cysticerciæ in pigs, echinococci and cœnuræ in cattle and sheep), abscesses, older encephalitic foci, tubercles, chronic pachy-meningitis with thickening of the dura, exostoses, etc. In its nature, therefore, this form is quite different from true epilepsy, and only agrees therewith in its symptoms; the spasms which it produces are consequently always spoken of as epileptiform, in contrast to the truly epileptic. But since the causes of symptomatic epilepsy can be known only after death, its spasms are also alluded to in life as epileptic fits.

3. *Traumatic epilepsy* starts from injuries of the cranium and brain; the epileptic fits often not occurring until long after infliction of the injury. The pathological process may therefore vary. Either we have a lesion of the cerebral cortex, a compression of it by blood, bone-splinters, etc., or a disease of the membranes of the brain. This form of epilepsy has been produced experimentally in guinea-pigs by blows on the skull, the disease setting in after some weeks (Westphal). It is also asserted that epilepsy thus traumatically caused by mutilation of the cortex of the cerebrum in dogs can be transmitted by heredity to their offspring.

4. *Reflex epilepsy* occurs in certain nervously disposed animals after the action of irritants upon their peripheral nerves. Among these are wounds and contusions of the peripheral skin-nerves, compression of the same by tumours, as well as irritation of the mucous membranes by parasites. Of the latter, ascaris megalocephala and mycotic otitis (aspergillus nigricans in the shell of the ear) are blamed in horses, and in dogs tape-worms and the pentastoma tænioides which occurs in the frontal sinus. Nervously disposed dogs are said to have epileptiform spasms in constipation, which may return again later: Injury to the mucous membrane of the mouth by sharp-edged

teeth in horses when eating may occasionally bring on an epileptic attack. Gerlach observed a horse which collapsed with epileptic spasms on being touched upon the withers, where it was very sensitive. Schrader saw another which fell into fits on touching a certain spot upon its skin in the middle of the left side of its neck. Brown-Séquard and others produced this reflex epilepsy experimentally in rabbits by injuries to the peripheral nerves. For instance, in consequence of section of the ischiatic the malady appeared in from 11 to 71 days, continued for some time and was occasionally even transmitted to progeny.

Anatomical Conditions.—As already stated, there are no pathological changes in the brain in true epilepsy. But in the other forms we find, according to their respective etiology, tumours, exostoses of the cranium, thickening, adhesion or ossification of the cerebral membranes, encephalitic foci, etc. Thus in an epileptic cow Voigtländer observed in the middle of the right hemisphere of the cerebrum a partial suppurative encephalitis in consequence of fracture of the horn. Cerebral œdema, chronic hydrocephalus and partial atrophy of the brain are also found. In nine epileptic horses Bassi found pronounced asymmetry of the skull. Siedamgrotzky also noticed in a dog that the skull-bones were distorted, lumpy and uneven, the cranial cavity and the brain were very small, and there was chronic hydrocephalus with dilatation of the ventricles and atrophy of the adjacent brain-parts (rickety changes).

Symptoms.—The characteristic epileptic fits occur either as general muscular spasms spread over the whole body and coupled with insensibility (complete, general epilepsy), or the spasms are restricted to single groups of muscles, especially of the head and neck, and during which the animals can retain their feet (incomplete, partial epilepsy). But transitions from one form to the other occur in the same animal. According to Unverricht, every epileptic attack consists of two parts. At first, the entire muscular structure of one-half of the body twitches in a typical manner, beginning at the hinder extremities, and then extends to the other half.

1. *Severe epileptic fits* (complete general epilepsy), as observed in the stable or when using the animals, occur sometimes very suddenly. Dogs and pigs, after staggering for a moment, suddenly fall senseless to the ground, often with loud cries,

and are attacked by violent tonic and clonic convulsions. There are usually prodromal signs, premonitions, which seem to point to loss of equilibrium, or dizziness. In rarer instances the attack commences, as in man, with peculiar disturbances of sensation on single limited spots upon the body, so-called sensible "aura." Thus Hertwig saw the attack begin in two horses by their endeavouring to scratch their heads with their off hind foot. The usual course of a fit is as follows:—Horses in the stall suddenly hang on the halter; those in movement stand dead still and display a certain excitement and dread. Dogs run about terror-stricken and often as though senseless, or stare around stupidly and shake their heads. In all animals the glance is staring and expressionless, the eyeball looks rigid and fixed, they begin to tremble, stagger to one side or a few steps back and straddle widely with the legs to support their tottering bodies. The convulsive twitchings which now set in generally begin at the head, in other cases in the neck, and consist in blinking the eyelids, rolling, twisting and even squinting of the eyeball, twitching, dragging movements of the muscles of the lips, nose, cheeks, and ears. To these spasmodic action of the jaws is added, whereby the abundantly secreted saliva is worked into a lather. The jerky withdrawal of the posterior maxilla occurs sometimes so violently that loud smacking noises are heard. In other cases the jaws are tightly pressed together, we hear grinding of the teeth, or again the mouth is kept wide open for some time. The spasms gradually pass on over the neck and trunk. Head and neck are stiffly bent in one direction, mostly to one side, the same also happens to the back, so that the head and neck may be drawn against the lateral thoracic wall. The animals consequently lose their balance and tumble down. While on the ground the convulsions attain their greatest severity and extend to the extremities. The animal struggles or beats convulsively with its feet, and complete loss of consciousness and of sensation soon follows.

The mucous membranes of the head, especially the tarsal conjunctiva, are pale at the beginning of such an attack, the pupils dilated and insensible to light, and reflex processes are mostly suppressed. The heart action is irregular and slow, the speed of the pulse sinks below half its normal rate, the pulse itself being small and hard. Breathing is strained while the animal lies, even panting and rattling. Dogs and pigs often yelp and squeal. In consequence of the dyspnœa and violent muscular action, considerable venous hyperæmia of the head

may set in towards the end of a fit ; outbreaks of sweat are also noticed, particularly over the elbows and in the flanks.

These convulsions sometimes cease suddenly, at others gradually, and may give place to a brief attack of general tetanic spasms, during which involuntary passage of fæces and urine is sometimes seen. When the attack is over, consciousness and sensation generally return quickly. Only in very severe cases does a visible indisposition, which may last up to an hour, remain behind. The animal rises from the ground and gradually recovers, manifesting for a time a certain lassitude and exhaustion. Dogs often get up as soon as the fit is over, shake themselves and recognise their surroundings ; but sometimes the attack is ended by a deep, snoring sleep. Some have observed as the immediate sequel of such fits an unusual and even maniacal excitement. Dogs will run aimlessly away, or show a tendency to bite. Schrader saw a horse which bit its own fore-leg after the spasms had ceased. Abortion occurred in one case, that of a mare, as the result of an epileptic attack (Eberhard).

2. *In slighter epileptic fits* (incomplete partial epilepsy), which are also accompanied by suspended consciousness, the spasms often remain localised upon certain muscles of the head, or they pass on over the neck and trunk, thus forming a transition between the slight and the severe. Frequently we notice only blinking, squinting or rolling the eyes, with spasmodic twitching of the facial muscles, especially of the lips. Such slight attacks in horses bear a great likeness to vertigo ; they occur particularly during movement and in horses with blinkers, but may also happen in the stable or directly a horse is taken out and harnessed. In stronger spasms the animal shows a backward twitching of head and neck, which are best seen sideways, and trembling of the fore-quarters (cows fall on their knees or lean against the wall). These in addition to the spasms in the head just named.

Duration and Course.—The *duration* of epileptic fits is various, being generally longer in the severe attacks. It varies from a few minutes (we saw one which only lasted fifteen seconds) to a quarter or half-an-hour. The return of the paroxysms occurs at irregular intervals. They may be repeated several times in a day, or may cease for months and even for more than a year. Sometimes they are more frequent at the beginning of the disease, sometimes during its later course,

The violence of the several attacks varies greatly, even in the same animal; and mild fits may continue at the same degree for many years without an increase of intensity. Between the fits, animals usually appear quite healthy; occasionally there is a permanent psychic depression, diminished intelligence, even dulness and imbecility, trained dogs have even been known to forget their tricks. These signs may also arise from a simultaneously developed chronic hydrocephalus or cerebral atrophy.

The course of epilepsy is extremely chronic, the disease maintains its ground for years and even for life, an early disappearance after a few months is exceedingly rare. It may be very easily confounded with other conditions, and yet with proper treatment epilepsy is sometimes curable. As such it does not cause death, excepting when severe injury, hæmorrhage on the brain, etc., is produced accidentally when falling, which however rarely happens.

Differential Diagnosis.—The diagnosis of epilepsy is mainly based upon its chronic course and upon the paroxysmal character of its muscular spasms, which are always accompanied by loss of consciousness. Simple though it may seem to diagnose the disease from the above indications, yet in practice it is not always so easy and certain. Epilepsy may sometimes be confounded with the following diseases.

1. With *eclampsia* and *acute cerebral spasms*, the so-called epileptiform or epileptoid fits, which latter occur as symptoms of various acute brain-diseases or infectious ailments, such as distemper in dogs, or as part-symptoms in certain poisonings, or as simple and quickly passing reflex phenomena (teething convulsions). The only distinguishing characteristic here is the acute course—the spasms are exactly like those of epilepsy and are combined with loss of consciousness—but this acute course is not always easy to prove; then again, reflexly excited sympathetic cerebral spasms may also assume a chronic course, if the reflex agent continue its operation for long, as, *e.g.*, with intestinal parasites. In the latter case differentiation is exceedingly difficult and diagnosis often impossible during life.

2. The slighter epileptic fits may also be confounded with *vertigo*. The decisive criterion is that convulsions do not occur in vertigo, but in such slight cases the convulsions may be so feeble as only to be noted on careful examination (in man

they are sometimes lacking entirely); on the other hand, the struggling movements of the feet in very severe attacks of vertigo, when the patients fall down, closely resemble convulsive spasms, and explains why, from the scientific standpoint, no clear border-line has been drawn between epilepsy and vertigo.

3. *Thrombosis of the posterior branches of the aorta* (iliac, lumbar and pelvic arteries) has often been mistaken for true epilepsy, owing to the sudden break-down of the hind-quarters. Diagnosis is secured by examination per rectum, by the peculiar kind of functional disturbance in the hind limbs (paresis), the absence of spasms, and the possibility of producing at any time the symptoms of thrombosis of the aortal branches by suitably moving and exerting the animal.

Therapeutics.—Apart from the avoidance of any known causes, we may, at the beginning of an attack, always lessen its violence and perhaps prevent the animal from falling down if we hold its head firmly, cover up its eyes and bend its neck energetically towards the opposite side from the paroxysm. Other remedies for shortening the fits, such as smelling drugs, inhalation of chloroform, sprinkling with cold water, etc., are not applicable to large animals, and in smaller ones are almost worthless.

Against the disease itself a number of drugs are in use, which are mainly effective as they reduce the enhanced irritability of the brain. Success has been attained most frequently with dogs. According to our own experience and that of others, bromide of potash and bromide of soda are particularly good remedies for dogs; we give 3 to 4 doses daily for a considerable time, viz., 8 to 15 grains dissolved in water. Various bromides can also be prescribed together in the form of so-called bromide-water: Potassium bromide and sodium bromide of each 2½ drams; ammonium bromide 1¼ drams, water 10 oz., from a teaspoonful to a tablespoonful to be given three or four times daily. To horses and cattle we give this along with other salts and with aromatic vegetable powders mixed with their food, in doses of 5 drams to 1½ oz. Of other remedies we might mention: nitrate of silver (dogs, ¼ to 1½ grains per day in three or four doses; horses, 15 to 30 grains); oxide of zinc (dogs, ⅜ to 3 grains); belladonna (atropin. sulph. $\frac{1}{15}$ to ⅛ grain for dogs and ⅛ to 1½ grains for horses); sulphate of copper, valerian root, etc.

ECLAMPSIA.

General Notes on the Definition of Eclampsia.—Under the title of eclampsia a number of diseases both of man and of animals are grouped together which, owing to their manifest differences, can hardly be brought under one scientific definition. In its widest sense the word eclampsia is used as a collective name for several convulsive conditions whose common characteristic consists in tonic and clonic spasms, which run acutely with or without unconsciousness. Under this very wide definition have been included a number of purely symptomatic spasms arising from well-known pathological changes, which do not merit the name of a special disease. Thus the cerebral spasms which occur during brain diseases, or the epileptiform spasms of distemper should not have a separate name of their own, nor those which arise as symptoms of uræmia or of certain poisonings, although at one time men spoke of an eclampsia uræmica or saturnina (lead-poisoning).

On these grounds the definition of eclampsia has recently been narrowed, and the name only applied to the cases of epileptiform spasms which, independent of certain organic diseases, manifest themselves as a pure neurosis in the form of an independent acute malady. According to this view, eclampsia would mean the same as acute epilepsy, in which the eclamptic convulsion passes off exactly in the same manner (and mostly in the way of reflex movement) as the epileptic, the two being only distinguished by their acute and chronic course. If we compare the several acute convulsive conditions recorded in our veterinary literature as eclampsia with this narrower definition, we shall find that an eclampsia identical with acute epilepsy does occur among our domestic animals. To this class belong especially the convulsions which are seen in new-born and very young animals (corresponding to eclampsia infantum), and certain descriptions of acute reflex spasms in full-grown animals. On the other hand, that which is commonly known as the " eclampsia of bitches " does not seem to belong to this form, because consciousness is usually unaffected, whereas in acute epilepsy there is a loss of consciousness. We have, nevertheless, followed the usual custom of treating the eclampsia of bitches in this chapter, as, owing to our imperfect knowledge of its nature, it is difficult to know where else to introduce it. There is no ground for describing paralytic

milk-fever as eclampsia, or for discussing it here, as we have more fully explained under the heading of Puerperal Fever.

1. **Eclampsia of Young Animals** occurs especially among teething dogs and sucking pigs. Convulsions are observed, very similar to the fits of children: twisting of the eyes, tremblings, jaw-spasms, grinding the teeth, foaming and salivation, twitchings of the body, aimless running about, shrieking and whimpering, etc. Many animals expire suddenly with a loud cry. The causes are unknown. As a rule, a nervous disposition is blamed, or certain peripheral nervous irritations (change of teeth, helminthiasis, gastric or intestinal catarrh). According to later investigations in man, the cause is supposed to be poisoning (multiple epithelial necrosis of the liver and kidneys, according to Schmorl) or infection (basilary embolism of the liver and kidneys, according to Gerdes). *Treatment* is the same as for epilepsy, and consists in the use of anti-spasmodic remedies, especially bromide of potash, urethane, sulphonal, and chloral hydrate. We may also try clysters of ether, decoction of valerian root, etc.

2. **Eclampsia of Full-grown Animals** is very uncommon, and its morbid aspect most frequently that of acute reflex epilepsy. Thus, many animals fall into acute epileptiform spasms on being touched upon certain wounded or inflamed spots—cows, for instance, on touching sensitive injuries at the root of the horns. In the same way the epileptiform spasms which are observed in dogs after irritation of the external ear-passages by ear-mites must also be regarded as eclampsia. It is also not unlikely that many cases of reflex epilepsy are only repeated attacks of eclampsia, although we must not forget the possibility of the latter changing into true epilepsy. Lustig describes epileptiform convulsions in a horse which was in the convalescent stage of a severe lung-affection. Albrecht, Lameris and others several times observed eclamptic fits in cows soon after calving, which had nothing to do with milk-fever and took the form of tetanic spasms in neck and head, grinding the teeth, unconsciousness, falling down, spasmodic movements of the limbs, twisting the eyes, etc. These fits lasted from a few minutes to half-an-hour, and when over the animals were generally quite well. The shortest duration of the ailment was 2 days, the longest 18 days. Similar attacks were seen in goats. The causes are here probably to be sought in inflammatory conditions of the uterus.

3. **Eclampsia of Suckling Bitches** (the so-called) is a disease whose origin is still very obscure. As it occurs mostly in bitches when giving suck, the act of suckling must as such stand in close etiological connection with it, so that we may presume an abnormal irritation, starting from the nerves of the milk-glands, to be its cause. More recent investigations (Friedberger, Druet) have, however, shown that the malady does not appear exclusively just after giving birth, but may occur some days before, or even some weeks after. In one case the spasms were seen eight days before parturition and in another 50 days after. We must then here presume a predisposition to spasms arising from the state of pregnancy or from having brought forth. Those which seem most inclined to the sickness are the smaller, tenderer breeds, also pampered animals, very intelligent ones, and those which yield much milk. As accidental causes, some blame strong emotion, such as grief for lost puppies, fear or excitement on being punished, and also cold. The Berlin dog-hospital reported 64 cases of eclampsia among 70,000 dogs treated in 9 years, as against 419 of epilepsy.

The *symptoms* generally appear in the interval between the second and thirtieth day after giving birth, and consist essentially in fits of tonic and clonic spasms, especially of the extensor muscles, the sensorium remaining fairly clear. The attacks come suddenly without noticeable warning. The dog shows a certain unrest, excitement and alarm, the latter being especially manifested in its eyes; breathing is quick, short and strained, as also are both pulse and heart-beat; the visible mucous membranes of the head are reddened, and sometimes after even a quarter of an hour a striking and often rapidly increasing weakness is observed in the hind-quarters. The animal is soon unable to stand, falls down, lies on one side with her feet stiffly stretched as though dead and in rigor mortis, so that the extremities can sometimes only be bent with considerable force, and the bitch, if placed on her feet, will stand like a saw-bench. Every independent motion of the limbs seems to be impracticable. At the same time brief convulsive tremblings of the limbs and also of the trunk occur, with distinct clonic spasms, which not rarely are very violent and affect especially the extensor muscles of the limbs, neck and back, but also occur in other places, such as the temporal muscles or those of the eye. With the appearance of the spasms, breathing becomes more rapid (up to 100 a minute) and at last throbbing, panting

and languishing, the mouth being wide open, often covered with foam, and the tongue hanging out. The heart is palpitating and its frequence increased to 160 beats a minute or more. Sometimes the jaws are convulsively opened and shut, the tongue drawn back and swallowing movements made. The redness of the mucous membranes of the head continually increases, the injection of the conjunctival vessels being particularly marked; even hæmorrhage in the mucous membranes may take place. The abdomen is often sensitive to pressure, eating and drinking are entirely relinquished, and also the passage of fæces and urine. After an attack the urine is found to contain albumen. At the outbreak the teats are very much swollen, very full of milk and high in temperature, but later on are mostly quite collapsed and empty. Consciousness and sensibility are in no way disturbed. The face, as Mauri very justly remarks, manifests fear rather than pain. The pupils are of normal size. External irritants cause no noticeable increase of the spasms.

The *course* of the disease is acute, often even peracute. The attacks generally last from one to two days, more rarely a few hours, the spasms do not continue with equal violence, there being intermissions during which the animals try to stand up, but are unable to use their hind-quarters. Finally, the convulsions give place to a sleepy condition. If this be then left to itself, death generally follows in one to two days, from paralysis of the brain or asphyxia. Proper treatment, however, regularly leads to recovery, usually in a very short time and even in an hour. We have occasionally seen instances of spontaneous recovery, but they were rare.

A *confusion* of the eclampsia of suckling bitches with strychnine poisoning is scarcely possible. The absence of an increased reflex irritability of the spinal cord, *i.e.*, reaction to external irritation, which absence marks this form of eclampsia, makes differentiation easy. It may also be known from epilepsy by its acute course, and from distemper by the absence of the other characteristic signs.

Treatment consists either in subcutaneous morphia injections (Siedamgrotzky), an average of ⅛ grain of morphium hydrochloride in 15 minims of water, or in cautious chloroforming up to the commencement of narcosis. We have also observed good results from urethane (1½ to 5 drams) and hypnone (4 to 30 grains). Mauri speaks well of chloral hydrate (8 to 30 grains); Trasbôt of ether (ether-syrup); and Zimdel recom-

mends chloroform-syrup (15 grains of chloroform to 3 oz. of syrup, a teaspoonful to be given four times at intervals of 15 minutes, and then after intervals of two hours). Possibly the favourable effect of these narcotics, which also dilate the vessels, may give us some hint as to the nature of the disease and allow us to trace it from a rapidly-developed and just as rapidly-corrected disturbance of circulation in certain cramp-centres. Marked pathological lesions are not present, and cannot be found by dissection. Possibly the ailment springs from anæmia of certain nerve-centres in the brain, set up reflexly by the nerves of the mammary glands or of the uterus. This theory is supported by the fact that in many cases of eclampsia we have been able to demonstrate by the ophthalmoscope a clearly marked anæmia of the papilla of the optic nerve. The beneficial effect of chloroform may perhaps be explained by assuming that it is an antidote to all poisons which produce spasms, and that here also a toxin must be held to represent the original cause of eclampsia (ptomatotetanin). These are, however, merely hypotheses, and more exact etiological and pathological investigation is greatly to be desired.

CATALEPSY.

Definition and Nature.—Catalepsy is a peculiar periodic morbid condition, characterised by contraction of the body muscles, whilst passive movements and changes of posture can be made with remarkable ease, consciousness and sensation being at the same time lessened or even quite suspended. Some hold that this cataleptic rigidity arises from a transient heightening of the normal tonicity of the voluntary muscles, and others attribute it to an abnormally increased resistance of conduction within the motor ganglia, whereby the excitability of the muscles is reduced by the will and by external irritants. The disease occurs in our domestic animals both as an independent primary affection, and also secondarily in the course of other nervous diseases in the form of so-called cataleptic fits. It seems to be extremely rare as an independent disease among our animals, and has hitherto only been noted in solitary cases, among dogs, horses, cattle and wolves. It is commoner in man, in whom it represents a kind of hysteria.

Etiology.—The causes of catalepsy are not yet known. Violent emotion is often blamed (terror), but this can only

be regarded as contributory. The chief factor must be sought in an as yet not clearly understood nervous predisposition; for the disease has many resemblances to the neurosis called hysteria in man.

Anatomical Conditions.—Positive pathological changes of the central organs are as conspicuous by their absence in catalepsy as in eclampsia or tetanus. The disease must rather be regarded as a purely functional neurosis of the brain and spinal medulla. Occasionally certain secondary changes of the muscles are found which have some resemblance to those occurring in tetany, especially hæmorrhage, opaque swelling and fatty degeneration of the muscles of the skeleton, waxy degeneration of the cardiac muscular fibres, and also hæmorrhages in stomach and bowels (Fröhner).

Symptoms.—The attacks generally occur suddenly, but occasionally certain warning signs are noticed, such as restlessness, excitement, change of appetite. The rigidity begins in the muscles which happen to be in action, but spreads very rapidly over all those controlled by volition. As the animals are thus deprived of all power of voluntary movement, they stand motionless, like statues, and as though charmed, in the posture in which the fit surprised them. The affected muscles feel tense and firm, and occasionally stand out as if chiselled. At first they offer more or less resistance to any passive change of attitude; but this grows gradually fainter, and it is then possible to bring the limbs into all kinds of postures, in which they will remain unchanged for a long time (so-called wax-like flexibility, flexibilitas cerea). Consciousness and sensation are more or less considerably disturbed. Fröhner observed a perfect ecstasy in a cataleptic dog, a hypnotic state strongly reminding one of the descriptions of somnambulism in man. The eyes are rigid, appear dull as though veiled, and their pupils are at first enlarged and then contracted to the size of a pin-head. During the attacks the sensibility of the skin and mucous membranes is diminished or completely paralysed, and so, too, are the organs of sight, hearing and smell. The urine passed immediately afterwards contains albumen, and also much biliary pigment. The circulatory and respiratory apparatus are normal. Hennig observed sudden rigor of the entire muscular structure in a horse, accompanied by complete loss of consciousness and sensation in the skin.

The *course* varies much in duration, and the attacks may recur. Spinola fixes their length at from a few minutes to an hour and longer; Hertwig speaks of them as lasting several weeks (?); Fröhner observed two attacks in a dog with a duration of 12 and 30 hours. The condition is not as a rule fatal, but Hertwig says that animals may die of exhaustion brought on by interrupted consumption of food and drink. Fröhner saw one dog die on the seventh day, after the cataleptic rigidity had been followed by relaxation of all the muscles, including the involuntary muscles of the bowels and bladder, with a fall of bodily temperature and increasing coma. In a case observed by Hennig, in a horse, a morphia injection was given and recovery followed in 1–24 hours

Therapeutics.—Respecting the treatment but little is known. The narcotics (morphia, bromide of potash) are purely symptomatic in their effect. Perhaps we might try to restore the disturbed reflex excitability by cold douches or the electric current.

CHOREA. ST. VITUS'S DANCE.

Definition.—Under the name of St. Vitus's dance, we understand in man continuous involuntary twitching movements of the body muscles, mostly irregular and seldom with any apparent purpose, which are complicated with more or less decided disturbances of the sensorium and of sensation. The disease has therefore been called muscular restlessness, muscular delirium or convulsions. The cerebrum has lately been assumed to b the seat of this neurosis, and capillary embolism of the corpus striatum and the thalamus opticus has been especially suspected as its cause, because chorea occurs in man with striking frequency along with acute articular rheumatism, endocarditis and valvular defects. The severance of the spinal cord in dogs suffering from chorea carried out so successfully by Chauveau and others seems to indicate that this organ also has something to do with the pathogenesis of the malady. How far the analogy between chorea in domestic animals and in man holds good is difficult to say with the small number of recorded cases before us and the manner in which many of them are described. But it seems as though simple cerebral and reflex spasms, perhaps even eclamptic and epileptic fits,

had often been confused therewith. The spasms in dogs during distemper are not choreic, but epileptiform; but a symptomatic development of St. Vitus's dance is sometimes observed after recovery from distemper.

Etiology.—The causes of chorea are unknown. Young, feeble and anæmic animals suffer most frequently; probably a nervous predisposition plays the chief part. The complaint is said to have been observed in horses, cattle, dogs, cats and pigs.

Symptoms.—From the reports of numerous observers it appears that the characteristic involuntary twitching movements are sometimes confined to the muscles of the head and neck (nodding, shaking the head), and again are spread over the entire muscular apparatus of the body (spasmodic motions, sometimes automatic or like a dancing puppet). Hering noticed in a horse continuous up and down movement of the head and of one fore leg; Leblanc, irregular twitchings over the whole body, whereby first one foot, then the head, the fore-quarters, the hind-quarters, an ear, a lip, an eyelid, etc., were moved. Anacker observed in a cow rhythmical movements of the head and neck from side to side, whereby the hind feet danced up and down as though walking. Schleg saw also in an ox continuous swaying to and fro of the head, the forelegs being thrown high up when walking. Hess and Verwey found clonic rhythmic spasms in young pigs over all the muscles of the trunk; some of them constantly flung their heads about, others kept making lifting movements with the rump, which was first raised into the air and then sunk to the earth. The *course* of St. Vitus's dance in our domestic animals appears to be chronic. Hess observed recovery in young pigs in from seven weeks to several months; and Leblanc noted a case in a horse lasting more than a year. The *prognosis* does not, however, seem to be unfavourable.

Therapeutics.—As in the other neuroses, the narcotics, chloral hydrate and bromide of potash are recommended; Legros and Onimus saw a choreic dog restored to quietness after a clyster of 50 grains of chloral hydrate. We can confirm these statements, but would remark that the sedative effect of narcotics is mostly only fleeting. Palombo says that he cured a dog by dipping it five or six times daily in cold water.

Oxide of zinc and arsenic are also used. The latter we administer in the form of Fowler's solution, usually for the chorea which remains behind after distemper: Fowler's solution and cinnamon water of each equal parts, give from 5 to 10 minims daily.

VERTIGO. DIZZINESS.

Definition.—Under the term of vertigo we describe periodical and suddenly recurring abnormal sensations respecting the relation of an animal to its surroundings, viz., an unusual feeling of movement, with a loss of the sense of equilibrium and consequent incapacity to remain erect. The nature of these morbid sensations is explained by functional or anatomical disturbance in the central organ for co-ordinated locomotion, *i.e.*, in the cerebellum. Vertigo is mostly only a symptom of other diseases and is but rarely a pure neurosis.

Occurrence.—Vertigo occurs most frequently in horses and dogs, more rarely in such animals as cattle, pigs or sheep. Full-blooded, well-nourished, and but little worked horses of medium to advanced age are most liable to attack. Spring seems to be among the seasons that in which vertigo is most rife; to which the prolonged winter sojourn in the stable, the rapid changes of temperature in spring, combined with the change of coat then in progress, all contribute. The condition is generally observed when the horse is in motion, more rarely in the stable, and usually only when being driven, much less often in riding. Some horses, indeed, which are habitually subject to vertigo in harness, are quite free from it when ridden. Many animals have regularly every year a few attacks of dizziness as winter passes away, and are free all the rest of the year. Among dogs the defect is noticed, especially in aged draught-dogs with heart-disease.

Etiology.—1. The causes of *independent essential vertigo* are, like those of all pure neuroses, unknown; there being no pathological changes in the cerebellum. In veterinary nomenclature this form has hitherto been known as "brain-dizziness."

2. Those cases may be termed *symptomatic vertigo* which can be traced to permanent intra-cranial pathological changes.

Among these belong tumours of the brain, of which cholesteatomata may be cited as a cause of the disease in horses, also encephalitic foci of softening, chronic inflammation of the cerebral membranes and affections of the cerebral vessels. Fugitive and non-periodic fits of dizziness are also observed in acute affections of the brain, *e.g.*, in acute inflammation, or in acute anæmia brought on by severe loss of blood, or by rapid emptying of fluid accumulations from the body cavities, or of gas from the paunch.

3. The so-called "congestion vertigo," caused by *chronic disturbance of circulation in the brain*, is the form oftenest observed. Various diseases of the heart, vessels and lungs, which are accompanied by a congestive hyperæmia of the lungs, lie at the root of the condition. This congestive hyperæmia is in its effect precisely the same as an arterial anæmia of the brain, and excites the cerebellum partly by a lack of oxygen and partly by excess of carbonic acid. Thus we observe fits of dizziness in dogs in the course of valvular defects, in horses also in consequence of chronic, degenerative lung diseases, aneurysms of the pulmonary artery, etc. They are also very often caused by compression of the jugular veins in the neck by collar or breast harness, or by a too tight throat-latch, or a too short bearing-rein (the latter particularly if combined with a tight bit). The operation of these causes is intensified by exhausting movement, great heat, warm and stuffy stables, full-bloodedness, etc. General chronic anæmia is also frequently accompanied by attacks of vertigo.

4. *Sympathetic* (consensual) or *reflex vertigo* arises, like reflex epilepsy, for which it is not seldom mistaken, through the transmission of a peripheral excitation to the cerebellum. The notoriously common "ocular dizziness" is included here. Among its causes are reckoned eye-blinkers with very shiny, smooth inner surfaces; blazing sunshine; strong reflected light from shining surfaces (lakes, rivers, pools, or snow when illuminated by the moon); rapid alternations of light and shadow in avenues of trees; as well as certain diseases of the eye, especially incipient amaurosis. Fits of vertigo are also observed after irritation of the ears, especially of the external passages, by foreign bodies; and in sheep during the disease caused by the larvæ of cœnurus cerebralis. Whether intestinal worms can produce vertigo, as some affirm, is still uncertain.

So-called *abdominal vertigo* is presumably caused by certain diseases of the abdominal organs, especially by chronic gastric catarrh and in-

testinal catarrh, engorgements in the portal venous system, and by liver complaints. Cases in which horses manifest dizziness when turning a windlass or gin, or when being transported by rail or ship, must be regarded as *physiological vertigo*. In animals, as in man, true sea-sickness takes place, especially in dogs, being rarer in horses. *Toxic vertigo*, *i.e.*, dizziness caused by poisonous food (equisetum, solanin, lolium temulentum, sedge-grass, alcohol) does not belong to this chapter really, and will merely be mentioned. The same applies also to the so-called *pasture-dizziness*, which is likewise certainly of toxic origin.

Symptoms.—The phenomena of vertigo are fairly simple. The horse suddenly shows a slower pace or stops entirely, makes shaking, nodding movements of the head, trembles, snorts, sways, staggers, straddles with its legs, presses backwards or sideways, holds its head up or on one side, leans against the carriage-pole or against the next horse (if in stable, against the wall), turns a few times round in a circle and then hangs in its harness or in the halter. It betrays fear and uneasiness, sweats frequently and, in severer cases, cannot stand upright, but, if driven further, tumbles over, when it lies quiet for some minutes and then struggles to its feet and, after a brief interval of lassitude, appears quite well again. During the attack consciousness is lost, sensibility of skin and mucous membranes is reduced or quite gone, the pupils are dilated, pulse and breathing quickened and occasionally fæces and urine are involuntarily passed: dogs frequently vomit. The *duration* of the individual fits is various, but is usually much shorter than in epilepsy, averaging from one to two minutes, in severer ones about five minutes.

The *differentiation* of vertigo from epilepsy is based mainly on the absence of spasms in the former.

Therapeutics.—The treatment of vertigo should be guided entirely by its causes, which we must first of all endeavour to remove. Thus many horses, accustomed to stand too much in their stalls, have been cured by daily use and hard work. If the cause can be traced to a certain form of blinkers, they must be removed or altered. According to Baker, shading the eyes with a piece of leather 2 to 4 inches broad, passing in front from one blinker to the other, has done good service. Straps which are too tight must be slackened, horses working a gin or other roundabout must be blindfolded. During an attack try to support the horse and cover up its eyes, if possible, take it out of the shafts, and see that everything be removed which

might damage it in case of a fall. If any cold water be at hand, the attack may be warded off or shortened by pouring it on the animal's head. Finally, as a prophylactic, occasional derivation to the bowels by laxatives is to be commended, but no blood-letting. The vertigo accompanying heart-disease should be treated with digitalis.

Menière's Disease.—Peculiar fits of dizziness in man arising from disease of the semi-circular canals of the inner ear (vertigo labyrinthina) are known by this name. Fleming has observed similar cases in horses. They manifest themselves in the sudden shaking of one ear with sharp inclination of the head to that side, lateral turning movements, excited looks and tumbling over to the same side. Consciousness remains undisturbed. In a few minutes after falling the horses get up again, but often continue for some time to be much excited. We have seen similar cases in horses. Sellmann has described one in a cat.

Basedow's Disease.—By this name we indicate in human medicine a neuropathic affection of the cervical sympathetic, which has not yet been etiologically cleared up, but which is characterised by three essential main symptoms genetically connected with each other.

1. Palpitation of the heart and vascular pulsation.
2. Swelling of the thyroid gland (struma).
3. Exophthalmus of both eyes (goggle-eye disease).

In many cases diseased changes in the lower cervical-ganglion of the sympathetic have been found as causes of the disease, and it is therefore supposed to arise from an inflamed condition of the sympathetic. Other observers presume a central origin (vaso-motor centre). The complaint is frequently complicated with other brain diseases (epilepsy, mental disease). In recent veterinary literature five cases of Basedow's disease have been reported, viz., two each in horses and dogs and one in a cow. Jeswejenke observed in a four-year-old full-blooded mare, after a race, palpitation, accelerated pulse, growing weakness, swelling of the thyroid gland, and also, sixteen days later, excessive exophthalmus of both eyes, whereby the eyes were so strongly forced outwards that the eyelids could no longer cover them. The horse died after an illness of four weeks. He saw a similar case in a seven-year-old pug bitch, but she was cured after being treated for three months with iodine. Röder described the condition in a cow fourteen years ago. Here also severe exophthalmus was observed with strabismus convergens, palpitation, quickened pulse, and also a struma as large as the fist. The disease had then lasted four years. Cadiot found in a horse hypertrophy of the heart and thyroid gland (there was no exophthalmus). Ries saw a case in a mare, where a goitrous growth, exophthalmus, and severe nutritional disturbance were all present. Interesting cases in dogs have been described by Albrecht and Lellmann.

CHAPTER XIX.

APPENDIX TO DISEASES OF THE NERVOUS SYSTEM.

CRIB-BITING.

Definition.—This may be regarded as a species of playfulness, or as a bad habit, and consists in involuntary gulping-in of the air, either alone or mixed with saliva worked into a froth. So far back as 1664, Solleysel correctly recognised crib-biting as such, when he compared it to smoking and taking snuff in men. It occurs oftenest in horses, but is not rare among cattle; one case has been observed even in pigs. Among horses the animals kept for purposes of luxury, thoroughbred racehorses, stud and cavalry horses show the vice most frequently, though ordinary working horses also practise it. The exact process in crib-biting is that, during a temporary interruption of breathing and visible contraction of the muscles lying on the anterior margin of the throat (sterno-thyroid, omo-hyoid, sterno-hyoid), the larynx and base of the tongue are drawn downwards, whereupon the upper part of the pharynx becomes filled with air, which, during the then following act of swallowing and return of the larynx and tongue-base to their former positions, partly escapes forwards and is partly swallowed, by which one or two so-called champing sounds are produced, *i.e.*, peculiar tones like belching. Either all or a part of the air may escape forwards, or the whole may be swallowed. The older view that crib-biting was, on the contrary, an eructation of gases from the stomach, like the belching of cattle, and was therefore always a sign of digestive disorder or anatomical changes in the stomach, has now been generally abandoned.

Dieckerhoff defines crib-biting as "a peculiar trick in inspiration, by which the head is drawn back towards the cervical vertebræ and either held or supported in that position; the thorax is then drawn forward and

filled; finally, air is taken in through the nostrils and into the pharynx and larynx. According to Dieckerhoff, crib-biting is not an act of deglutition, and the sounds are not caused by the swallowing of air, but by the passage of air into the larynx. Malkmus has, however, shown by experiment (pneumograph) that Dieckerhoff is wrong. He believes that the action is performed in the following manner: As the head is thrown forward, the larynx is drawn down; the pharynx is opened with a jerk and the air rushes into it, giving rise to the characteristic grunting sound. In the forensic aspect of crib-biting, it is necessary that both the noise and the drawing-down of the larynx should be established.

Etiology.—The causes of crib-biting vary.

1. The vice may develop *spontaneously* if the animals grow weary of long standing and idleness in the stall—the parent of vice. The animals endeavour to while away their time by licking the manger, crib, stalls, by biting all the wood-work within reach, by playing with their chain and its weights, etc., and this more especially when tied up during the day. Other habits are also formed at the same time, such as sharpening the teeth, swallowing saliva and so forth. In particular the playing and striking with the lips, and sucking and swallowing air and saliva lead frequently to what we call crib-biting. If horses are kept regularly at work, they never think of such things, and have, in fact, no time for them. Thus Pansecchi relates concerning the horses of a regiment in which the vice was widely spread, that after they marched out to the war of 1866 only one horse retained the habit, but that on return to garrison it became as common as before.

2. In addition to its spontaneous appearance, it is certain that *imitation* has much to do with its spread; young horses in particular soon learn the vice from their neighbours, and it may thus pass through all the horses of a regiment, stud or stable. It spreads even among older animals by imitation, though not so rapidly, and a horse eighteen years old was observed to begin crib-biting after standing for a time alongside a crib-biter.

3. That the evil may be *inherited*, especially from the sire, has been fully demonstrated. Thus Collin reports that the 45 descendants of an Anglo-Norman stallion were all crib-biters, viz., one foal at 3 months old, two at 7 and 8, five at from 10 to 12 months, the majority in their second and third years, and one after 3 years.

Among other causes, too energetic grooming has been blamed, or unduly tightening the girth, both of which make the horse very restless, causing it to bite the crib with all its strength and to cling fast to it; also high feeding with but very small supply of water (training racers and

hunters), which makes the animal try constantly to lick the manger, stall-sides, etc. (Sing). Hertwig's suggestion that the habit of crib-biting is preceded by a feeling of discomfort in the bowels or a slight indigestion, has not much probability in its favour; for the chronic changes sometimes found in the stomachs of crib-biters on *post mortem* (dilatation of the stomach, thickening of its walls, chronic catarrh) are much more likely to be the results than the causes of the habit.

Symptoms in Horses.—There are several kinds of crib-biting to be distinguished. 1. The commonest form is that of *crib-setting*, in which the incisor teeth, and sometimes those only of the upper jaw, or those of the lower jaw, or even of both, are placed upon the edge or bottom of the manger. But this does not form the only support; the bars of the hay-rack, halter, halter-chain, straps, carriage-pole, even the animal's own fore-leg, knee or hoof, are all used to rest the jaw upon. Günther saw foals use their mothers' hock-joints for crib-biting. In rarer cases the chin or the top part of the throat is laid on the manger instead of the teeth. 2. *Crib-biters* seize with their incisor-teeth any object, such as the edge of the manger, a nail, etc. We saw a horse which laid hold of the wall of the stone barrier as far as it could open its mouth. The essential phenomena then in crib-setters and crib-biters are that the animals press against the point of support, open their mouths as for eating, bend back their necks, stretching tightly the muscles on the anterior edge of the neck so that they stand out in knobs, and then emit one or two belching sounds. Thereupon they let go the point of support, and the act is ended. 3. The so-called *air-snappers* or *wind-suckers* perform the action freely without resting the head on anything. They stand back from the crib, bend the head down upon the breast, make certain peculiar movements with the lips as in taking food, jerk the head and neck quickly upwards and snap air in with the mouth, when a loud tone is heard. Sometimes we see repeated snapping of air with an ever deeper sinking and forward movement of the head (like a swimming dog making several vain attempts to secure a floating stick).

The earliest signs are generally playful movements of the lips and tongue and especially an increasing desire to lick and to swallow saliva. Crib-biting is occasionally only done at certain times, and again with extraordinary frequency, and by some animals at every hour or even minute, indeed every four or five seconds. But various animals show great differences. Some only do it in their stalls or when they believe themselves unob-

erved, others during every unoccupied moment, others again only just before feeding, some immediately after, while some do it only while eating or chewing, and when swallowing their food very rapidly. The most serious cases are those of crib-biters which practise the vice until they are quite blown out with air. During sickness or while living under very unusual conditions, the practice is generally discontinued.

The disadvantages of crib-biting are first, that much fodder is wasted, and that consumed is but poorly made use of, and secondly that neighbours are disturbed and may be contaminated by evil example. True signs of illness are very often absent. In other cases crib-biting is the cause of chronic disorders of digestion and nutrition, of tympanitic inflation of stomach and bowels, and of colic; on these grounds the habit has an essential pathological connection with digestion and its entire apparatus. As further consequences of crib-biting there has been observed (by Hell, Hartenstein, Röder and others) in animals which had brought the vice to a certain point of perfection an invariable hypertrophy of the sterno-maxillaris muscles and of the omohyoid muscles (caused by over-exercise).

Symptoms in Cattle.—The habit occurs here, too, in variously modified forms, but shows itself most frequently as follows:—The animal opens its mouth widely, raises the head, stretches out its tongue and persistently swings it to right and left, thereby beating its saliva into foam, which collects in the corners of its mouth and not unfrequently smears its outer cheeks; finally, with a clucking noise this is swallowed along with air. By this process, oft-repeated, the paunch becomes exceedingly and tympanitically inflated, when the practice is suspended and the air gradually ejected by repeated belchings.

That crib-biting may assume a different form in cattle is proved by a case reported by Weinmann, in which an ox stood still with close-shut mouth and constantly sucked in air through the corners with a peculiar wheezing sound, whereby its paunch became inflated like a drum, when the sucking ceased, and the air gradually came back by eructation. Johne also observed a cow which jammed her head firmly into the corner of the food-trough and by means of her tongue, and to the accompaniment of a singular clucking noise, pumped herself full of air, when she stood quite still for thirty or forty seconds and then audibly ejected the air in an unbroken stream and with evident signs of satisfaction.

In the Dorpat hospital crib-biting was observed among *pigs*, which placed their incisor-teeth, just like horses, on the edge of their food trough and emitted a similar sound.

Diagnosis.—A just recognition of crib-biting is of especial forensic importance, because in some countries it is one of those defects against which warranty can be legally demanded (in Bavaria, Württemberg, Baden and Hesse the period of warranty is eight days). Diagnosis is mainly based on proof of the muscular contraction on the anterior border of the neck and on the characteristic crib-biting sound. If air be swallowed, we may demonstrate the fact, if familiar with the animal, by auscultation or by compression of the pharynx with the hand on two different spots, as in the latter case a downward gliding, wave-like movement is felt. A wearing-down of the teeth may also occur in animals which are not crib-biters. Confusion with other forms of playfulness in which air is inhaled must be avoided; such are simple biting of the crib, sharpening the teeth thereon, saliva-swallowing, blowing with the lips (trumpeting), licking, striking with the mouth, etc.

Therapeutics.—As a prophylactic, isolation of the affected animals should be first attended to. Complete cure is possible at the beginning of the evil, but inveterate crib-biters are incurable. The following remedies have been tried:—

1. Intimidation by punishment; result doubtful.

2. Exhausting labour, avoidance of prolonged inactivity; result good.

3. Removal of all objects on which the jaws, etc., can be laid, tying up the animal, reversing it in the stall; result generally not very enduring.

4. Placing the manger on the ground; use of a nosebag; result doubtful.

5. Introduction of movable mangers, covering them with sheet-iron, fixing spikes on the edges and bottom of the trough; result doubtful.

6. A closely-fitting muzzle; result at first good.

7. Various head-straps, either in the form of a simple, narrow leather band, drawn tightly round the neck in the region of the larynx (which, however, compresses the blood-vessels too straitly), or broader straps with metal tongue, elastic steel clasp and sharp spikes (Ringheim and Burdajewitz's head-straps). But the latter may produce phlegmonous swelling of the neck as the result of injury inflicted.

8. Günther's crib-biters' pipes, which are hollow, perforated metal pipes inserted as mouth-pieces. Also various crib-biters' halters.

9. Severance of the omo-hyoid (Gerlach) or sterno-maxillaris muscles (Hertwig, Hell), or of both sterno-hyoid, or of both sterno-thyroid muscles (Dieckerhoff); but, according to the reports of Hering, Bassi, Vogt, Schwendimann, and others, the result here also is not durable. This is proved by numerous experiments undertaken in 1897–8 in the Prussian army. In the greater number of cases the operation was unsuccessful.

JIBBING.

Definition.—Jibbing is a vice of horses consisting in persistent, obstinate and conscious refractoriness against the requirement of an ordinary duty, and it not only greatly reduces the animal's usefulness but constitutes a general danger. We may distinguish between absolute and relative jibbing. The former means that the animal is useless, for all purposes, for driving as well as for riding. The latter, which is much commoner, is disobedience only in certain kinds of work. This detestable vice is far more common among harness than saddle horses.

Etiology.—The cause of jibbing is usually to be found in improper or brutal treatment of the animal when being broken in and trained to ride or drive, or in some senseless and cruel punishment. To these influences high-spirited and well-bred animals are very susceptible. Malicious teazing and irritation, as well as recovery from painful skin-injuries may give rise to jibbing. Among temporary causes ruttishness in mares must be named. How far heredity or pathological conditions of the brain may conduce to jibbing is not with certainty known.

Symptoms.—The phenomena of jibbing are partly active and partly passive, but both are frequently combined.

1. In *active jibbing* the animal stands still when at work and offers active resistance to all attempts to make it go forward: It skips here and there, presses to one side, throws itself on its fellow horse, rears, leaps over the traces, kicks, so that frequently harness, pole, and the front of carriage or cart are broken, even flings itself on the ground and strikes out with all four feet. Saddle-horses try to throw their riders, rearing and

bucking, or seek to knock him off against trees, walls, etc. Not unfrequently they fall over in their attempts. At such times horses manifest great excitement, a fiery eye, palpitation, reddened mucous membranes, trembling, quickened breathing and sweatings. In rarer cases this refractoriness increases to perfect mania, so that at last the animals are led away in a half-stupid condition. Great exhaustion generally follows on such attacks. Such animals usually appear quite good-tempered when in the stable.

2. In *passive jibbing* the animals while at work suddenly stand as though rooted and spellbound, back into the harness and stop the carriage intentionally, and cannot by any manner of means, kind or severe, be got to move a step forwards. Mostly they attempt to turn back. If left to themselves they usually go on of their own accord after a certain time. In other cases the driver must get down and unharness the animal, or else return home.

These two forms of jibbing may be variously combined. Very often the animal will obey if its load be lightened, or on the way back to the stable; but if heavily laden or in going up-hill, etc., will continue refractory.

Therapeutics.—Confirmed jibbers are incurable. Only in slighter cases, that is, during the development of the vice, can we obtain improvement, and this solely by psychic influences, by goodness and kind words, above all, by patience, but never by compulsion. In general, jibbing is regarded as incurable, although it may occur that jibbers appear to lay aside the vice for a while under well-accustomed surroundings. Ovariotomy is said to effect a radical cure in *nymphomania* in mares (Schwendimann and others), but the operation is frequently unsuccessful. Pilocarpine has by no means proved a reliable sedative in the treatment of obstinate cases in horses.

NERVOUS DISEASES OF POULTRY.

1. **Hyperæmia of the Brain** and **Hæmorrhage of the Brain** (apoplexy) occur among male birds, especially at the breeding season, after mechanical injury, as well as among young poultry if exposed to burning sunshine. Friedberger also noticed them in the course of chicken-diphtheria. The *symptoms* are dizziness, staggering, stupor, dilated pupils, rotary movements, motion backwards, irregular action of wings and feet, as well as peculiar epileptiform spasms, which are often caused by a mere touch. The head and neck are often so twisted that when the bird is

standing its skull is turned to the ground and the beak directed forwards. The creatures collapse, make straddling movements of the legs and twitch their wings, so that the prostrate birds are turned round on the ground. They then stand up again, unless the attack end apoplectically. As a *therapeutic*, cold is recommended and also purgative (for fowls a tablespoonful of castor-oil, or 1½ grains of calomel, or 8 grains of jalap root) (Zürn).

2. **Epilepsy** seems not to be at all rare in poultry. We have seen several cases in the different kinds of birds. In a case observed by Fried berger a goldfinch made flapping movements with outspread wings on the floor of its cage, had violent clonic spasms of the muscles of its ex tremities, fell on its back, or remained sitting on one spot with straddled legs, its head being spasmodically jerked up and backwards, its bill and eyelids opened and shut and its eyeball rolled, whereupon consciousness quickly returned. Bromide of potassium is to be used here, as for other animals (a 1 per cent. solution as drinking water).

END OF VOL I.

(For Index see end of Vol. II.)

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